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THE RELATION OF CERTAIN SPECIES OF PHYSALIS TO THE OVERWINTERING OF THE MOSAIC DISEASE OF CUCUMBER

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WITH PLATES XXXI AND XXXII

INTRODUCTION

A knowledge of the means by which the virus of mosaic diseases of cultivated annual plants overwinters is essential in developing control measures. It is known, in the case of the cucumber and the tomato, that certain perennial plants carry the mosaic disease over winter, and that insects may transmit the disease from these plants to the cultivated host in the spring. In the case of cucumber mosaic, a number of plants have been found to act as sources of primary infection, particularly the wild cucumber, *Micrampelis lobata*, (Michx.) Greene (8, 10), the milkweed, *Asclepias syriaca* L., and the pokeweed, *Phytolacca decandra* L. The wild cucumber is an annual, but it has been shown that the mosaic disease of this plant is carried over winter in the seed. The other two plants are perennials. The present paper deals with studies of the overwintering of cucumber mosaic on perennial species of *Physalis*.

For several years, experiments on the control of cucumber mosaic have been in progress at Madison and Rockland, Wisconsin, and Marengo, Illinois, under the direction of Dr. S. P. Doolittle. During these experiments, all the known and suspected wild hosts of the disease were removed around the experimental cucumber fields in an attempt to control the disease by eliminating the sources of primary infection. While assisting in this eradication work at Rockland, Wisconsin, during the summer of 1922, the writer noticed that numbers of mosaic ground cherry plants, *Physalis pubescens*

[1] The writer is indebted to Prof. L. R. Jones, of the Department of Plant Pathology of the University of Wisconsin, and to Dr. S. P. Doolittle, of the Office of Cotton, Truck, and Forage Crop Disease Investigations, for advice and suggestions during the progress of this work.

L. (Pl. XXXI, B), occurred near four of the most severely diseased cucumber fields. In view of the earlier work on the overwintering hosts of cucumber mosaic, it was suspected that certain species of *Physalis* might also be concerned in the overwintering of the disease, and a study was therefore made of the mosaic found on the cultivated ground cherry.

REVIEW OF LITERATURE

The first mention of mosaic on *Physalis* spp. was made in 1914 by Clinton (5), who infected two out of seven plants of a wild species of *Physalis* with expressed sap of mosaic tobacco plants. Clinton failed to mention the specific name of the *Physalis* used or to state whether it was an annual or a perennial.

Allard (1), in 1914, obtained infection on two garden species of *Physalis*, which he does not specifically name, with material from mosaic tobacco plants.

Nishimura (14), in 1917, found *P. alkekengi* L. to be a carrier of tobacco mosaic, although the plants showed no symptoms of the disease.

Crawford (6), in 1921, mentions in an abstract the fact that *Physalis longifolia* Nutt. is an overwintering agent of tomato mosaic.

Gardner and Kendrick (11, 12, 13), 1921 and 1922, who have done much work on species of *Physalis* in relation to the mosaic disease of tomato, have noted the occurrence of a mosaic disease on both annual and perennial species of *Physalis* in the neighborhood of tomato fields, as well as on several other solanaceous weeds; and they have proved the importance of the species *P. subglabrata* and *P. heterophylla* in connection with the overwintering and dissemination of tomato mosaic.

EXPERIMENTS WITH PHYSALIS PUBESCENS

Cross inoculations from Physalis pubescens to cucumber. The finding of mosaic plants of *P. pubescens* L. in the vicinity of badly infected cucumber fields near Rockland, Wisconsin, in the summer of 1922, led the writer to take specimens of the diseased ground cherry plants to Madison, where inoculations were made from them to healthy cucumbers. Crushed leaf tissue of mosaic plants was inserted in healthy plants by means of a sterile scalpel. These inoculations produced no infection, but inasmuch as the method used had proved less satisfactory than inoculation by means of aphids, in work with other mosaic diseases, it was planned to continue the work by use of these insects. Upon the next trip to Rockland, young ground cherry plants, both healthy and diseased, were dug up, taken to Madison, Wisconsin, and transplanted in the greenhouse. Aphids (*Aphis gossypii*) from a stock kept in the greenhouse on healthy cucumbers under cheese-

cloth cages were colonized on the mosaic *Physalis* plants, and after twenty-four hours were transferred to healthy young cucumbers under similar cages. Inoculations made in this manner produced infection in a high percentage of cases, as shown in table 1. Later, infection was obtained in a few inoculations made by the artificial method, but the percentage of infection was lower than when aphids were used. (See table 2.)

During these inoculation experiments it was found that the incubation period of the mosaic disease of *P. pubescens* ranged from five to fourteen days. Infection occurring more than fourteen days after inoculation was considered as accidental, unless doubtful symptoms had appeared before this time and later had become more definite.

TABLE 1.—*Results of inoculations of cucumber plants by means of aphids from mosaic and healthy plants of Physalis pubescens*

Date inoculated	Source of aphids	No. aphids per plant	No. plants inoculated	No. plants diseased	Date observed
	P. pubescens				
Aug. 22, 1922	Mosaic	15	12	6	Aug. 30, 1922
Aug. 22, 1922	Healthy (Control)	15	12	0	Aug. 30, 1922
Aug. 25, 1922	Mosaic	15	8	8	Aug. 30, 1922
Aug. 25, 1922	Healthy (Control)	15	8	0	Aug. 30, 1922
Mar. 31, 1923	Mosaic	20	8	7	April 5, 1923
Mar. 31, 1923	Healthy (Control)	20	8	0	April 5, 1923
April 4, 1923	Mosaic	20	10	6	April 12, 1923
April 4, 1923	Healthy (Control)	20	6	0	April 12, 1923
April 20, 1923	Mosaic	11	1	1	April 28, 1923
April 23, 1923	Healthy (Control)	15	1	1	May 1, 1923
Jan. 18, 1924	Mosaic	12	8	3	Feb. 1, 1924
Jan. 18, 1924	Healthy (Control)	12	4	0	Feb. 1, 1924
Jan. 22, 1924	Mosaic	30	15	5	Feb. 4, 1924
Aug. 5, 1924	Mosaic	20	10	5	Aug. 11, 1924
Aug. 5, 1924	Healthy (Control)	20	10	0	Aug. 11, 1924

Cross-inoculations from cucumber to Physalis pubescens. During the winter, the mosaic *Physalis* plants, which were at first thought to be perennials, died, and inoculations were made from mosaic cucumbers to healthy ground cherry plants. The results of these inoculations, set forth in tables

TABLE 2.—*Results of artificial inoculations of healthy plants of Physalis pubescens with crushed leaf tissues from mosaic and healthy plants of the same species*

Date inoculated	Source of inoculum	No. plants inoculated	No. plants diseased	Date observed
	P. pubescens			
April 21, 1923	Mosaic	8	3	May 3, 1923
April 21, 1923	Healthy (Control)	8	0	May 3, 1923
March 19, 1924	Mosaic	7	4	March 28, 1924
March 19, 1924	Healthy (Control)	7	0	March 28, 1924

3 and 4, indicate that cucumber mosaic may be transmitted to healthy ground cherry plants either by direct inoculation with aphids or by artificial inoculation. It will be noticed, however, that a much higher percentage of infection resulted when aphids were used as a means of inoculation.

TABLE 3.—*Results of artificial inoculations of healthy plants of Physalis pubescens with crushed leaf tissues from mosaic and healthy cucumber plants*

Date inoculated	Source of inoculum	No. plants inoculated	No. plants diseased	Date observed
	Cucumbers			
April 2, 1923	Mosaic	10	1	April 18, 1923
June 14, 1923	Mosaic	12	3	July 2, 1923
June 14, 1923	Healthy (Control)	12	0	July 2, 1923
Aug. 6, 1924	Mosaic	9	5	Aug. 23, 1924
Aug. 6, 1924	Healthy (Control)	9	0	Aug. 23, 1924

Cross-inoculations from pokeweed to Physalis pubescens. Pokeweed, *Phytolacca decandra* L., has been shown to have a mosaic disease identical with that of cucumber (9, 10). Aphids were colonized on mosaic pokeweeds and then transferred to healthy ground cherry plants. The results of these inoculations, given in table 5, indicate that pokeweed mosaic may be transmitted to *P. pubescens*, but the percentage of infection was lower than in the case of inoculations from the cucumber to *Physalis*. This may be accounted for in part by the fact that it is harder to colonize the cucumber aphis on pokeweeds than on the ground cherry. The symptoms produced in this case were identical with those resulting from inoculations from mosaic cucumber plants.

TABLE 4.—*Results of inoculation of healthy plants of Physalis pubescens by means of aphids from mosaic and healthy cucumber plants*

Date inoculated	Source of aphids	No. aphids	No. plants inoculated	No. plants diseased	Date observed
	Cucumbers				
March 26, 1923	Mosaic	25	8	8	April 4, 1923
March 26, 1923	Healthy (Control)	25	8	0	April 4, 1923
March 27, 1923	Mosaic	25	8	7	April 4, 1923
March 29, 1923	Healthy (Control)	25	8	0	April 4, 1923
April 8, 1924	Mosaic	25	10	4	April 17, 1924
April 8, 1924	Healthy (Control)	25	10	0	April 17, 1924
Aug. 25, 1924	Mosaic	25	12	7	Sept. 2, 1924
Aug. 6, 1924	Mosaic	25	12	11	Sept. 23, 1924

TABLE 5.—*Results of inoculations of healthy plants of Physalis pubescens by means of aphids from mosaic and healthy pokeweed plants*

Date inoculated	Source of aphids	No. aphids	No. plants	No. plants diseased	Date observed
	Pokeweed				
March 7, 1923	Mosaic	12	8	3	March 26, 1923
March 7, 1923	Healthy (Control)	12	8	0	March 26, 1923
March 21, 1923	Mosaic	20	8	1	April 4, 1923
March 21, 1923	Healthy (Control)	20	8	0	April 4, 1923
March 24, 1923	Mosaic	20	6	3	April 4, 1923
March 24, 1923	Healthy (Control)	20	6	0	April 4, 1923

Cross-inoculations from mosaic tobacco and tomato plants to Physalis pubescens. Aside from the pepper, *Capsicum annuum* L., no plant of the Solanaceae was known to be as highly susceptible to cucumber mosaic as *P. pubescens.* In order to determine whether this host was equally susceptible to tomato and tobacco mosaic, typical mosaic diseases of that family, inoculations were made from mosaic plants of these species to *Physalis.* The results of these inoculations appear in table 6 and show that *P. pubescens* is very susceptible to tomato and tobacco mosaic.

Physalis pubescens, an intermediate host by which tomato mosaic may be transmitted to cucumbers. The mosaic disease of tomato is identical

TABLE 6.—*Results of artificial inoculation of healthy plants of Physalis pubescens with crushed leaf tissues from mosaic and healthy tomato and tobacco plants*

Date inoculated	Source of inoculum	No. plants inoculated	No. plants diseased	Date observed
March 20, 1923	Mosaic tomato	8	6	April 1, 1923
March 20, 1923	Healthy tomato (Control)	8	0	April 1, 1923
April 7, 1923	Mosaic tomato	8	7	April 25, 1923
April 7, 1923	Healthy tomato (Control)	8	0	April 25, 1923
April 15, 1923	Mosaic tomato	16	15	May 2, 1923
April 7, 1923	Mosaic tobacco	8	8	April 20, 1923
April 7, 1923	Healthy tobacco (Control)	8	0	April 20, 1923
April 2, 1924	Mosaic tobacco	10	9	April 18, 1924

with that of tobacco, but it does not appear to be directly transmissible to cucumbers. To determine the relation of tomato mosaic to cucumber mosaic, both of which are transmissible to *P. pubescens*, inoculations were made from ground cherry plants, infected from mosaic tomatoes, to healthy cucumbers. This phase of the work is not extensive, but the inoculations made indicate that the ground cherry acts as an intermediate host by which tomato mosaic may be transmitted to the cucumber. In these experiments, aphids from a healthy stock kept in the greenhouse were colonized on mosaic ground cherry plants infected from mosaic tomatoes, and after twenty-four hours were transferred to healthy cucumber plants. The results of these experiments appear in table 7.

These experiments are analogous to those made by Doolittle (7) with pepper, a plant having a mosaic disease apparently identical with the

TABLE 7.—*Results of inoculation of healthy cucumbers by means of aphids from plants of Physalis pubescens infected with tomato mosaic*

Date inoculated	Source of aphids	No. aphids	No. plants inoculated	No. plants diseased	Date observed
April 20, 1923	Mosaic *P. pubescens* infected from mosaic tomatoes	20	1	1	May 1, 1923
July 13, 1924	do	25	4	2	July 20, 1924
July 13, 1924	do	25	3	1	July 20, 1924
Aug. 25, 1924	do	16	12	4	Sept. 2, 1924
Aug. 25, 1924	Healthy *P. pubescens* (Control)	16	6	0	Sept. 2, 1924

mosaic diseases of tomato and tobacco, and susceptible to cucumber mosaic. He found pepper to be an intermediate host through which cucumber mosaic might be transmitted to tobacco, and *vice versa*.

Symptoms of mosaic on Physalis pubescens. Although the mosaic disease manifests itself in slightly different ways on the different species of *Physalis* studied, the general characters are the same, and the mosaic symptoms on the different species vary in intensity rather than in nature. The cause appears to lie in the individual growth habits of the different species, since the symptoms vary greatly within a single species when the plants are grown during different seasons of the year, and the variations of symptoms within the genus is often no wider than in the species. The symptoms here described for *P. pubescens* are therefore essentially the same on the perennial species.

The mosaic disease first becomes manifest as a faint yellow flecking of the young leaves, an unusually decided case of which is shown in Plate XXXI, A. The mottling in this plant is of the above type, although the plant had been mosaic for some time. In other cases the symptoms consist of a general yellowing of the tip leaves accompanied by savoying and curling. This symptom, however, is always followed by a mottling of more or less striking degree. Under certain conditions, no decided symptoms can be recognized for a considerable time after inoculation, but eventually a few sharply defined, blister-like patches of deep green will appear in some of the younger leaves, although not necessarily in the youngest. There may be but one or two of these dark green areas on the leaves, but the diagnosis is reliable, for the plant will later show more pronounced mosaic characters, such as increased mottling, crinkling, or dwarfing (Pl. XXXI, B).

As the disease progresses there may be either increased or decreased mottling. When dwarfing is most pronounced there may be little mottling, but dwarfing is usually accompanied by striking malformations such as filiform and crinkled leaves (Pl. XXXI, B). In leaves of dwarfed plants the veins often stand out more distinctly than in normal plants. This apparently is due to the failure of the spaces between the veins to enlarge, leaving a much larger proportion of veins to leaf area than in healthy plants.

Under adverse growing conditions, symptoms are much less pronounced than under the most favorable conditions. Unless marked stunting and malformation has already taken place, mottling may disappear, leaving plants that are unthrifty in appearance but devoid of the usual mosaic symptoms, thus making it quite difficult, or often impossible, to diagnose the disease as mosaic. This condition is especially noticeable toward the latter part of the summer or in the greenhouse in midwinter. A faintness of symptoms, or their actual fading out, has been mentioned by other work-

ers in connection with ground cherry as well as with other plants. Gardner and Kendrick (11) mention the fact in their work with species of *Physalis*, while Allard (4, 2) notes it on tomato, *Nicotiana glauca* and *Datura stramonium*, and Clinton (5) on the tomato. The writer (10, 15) has described a similar condition on *Phytolacca decandra* and *Nicotiana glutinosa*.

It is possible that Nishimura (14) was unable to discern symptoms on *P. alkekengi*, since his inoculations on this species were made after the first of July. The existence of carriers of mosaic diseases that do not show symptoms at some time during their growth seems doubtful. The cases of *Nicotiana glauca* (3) and *Nicotiana glutinosa* (15) may be given as examples of plants once thought to show no symptoms of tobacco mosaic though serving to carry the disease, which have since been shown to be susceptible and to show definite symptoms of mosaic.

Relation of Physalis pubescens to the overwintering of cucumber mosaic. During the winter of 1922, seed collected from mosaic ground cherry plants at Rockland, Wisconsin, was planted in the greenhouse at Madison. These trials gave no indication that the disease was seed borne. A total of 1031 seedlings was grown from the seed of mosaic plants, but none of the plants showed any evidence of mosaic. It is probable, therefore, that the cultivated annual ground cherry is not a factor in overwintering of cucumber mosaic.

EXPERIMENTS WITH PERENNIAL SPECIES OF PHYSALIS

Although the cultivated ground cherry apparently was not concerned in the overwintering of mosaic, it was known that certain perennial *Physalis* species were affected with mosaic. A study therefore was made of all the plants of this genus which could be found in the vicinity of the experimental fields at Rockland, Wisconsin. In preceding years, wild cucumbers and milkweeds were found in considerable numbers at Rockland, many of them mosaic; but, as a result of the earlier eradication of these hosts, no wild cucumber and but few milkweeds were present during 1923. The discovery of large numbers of mosaic plants of two wild perennial species of *Physalis*, *P. heterophylla* Nees (Pl. XXXII, B) and the more abundant *P. subglabrata* McKenzie and Bush (Pl. XXXII, A), indicated the possibility of their being an important factor in the overwintering of the mosaic disease of the cultivated ground cherry and the cucumber, since the annual ground cherry was known to be susceptible to cucumber mosaic. These mosaic perennials apparently were limited to the neighborhood of fields then planted to cucumbers or were on land that had been in cucumbers at some time previously. Efforts were made to transfer roots of plants of both wild species to the greenhouse, but in most cases the plants died. Seed of both species were obtained from outside sources, but it was found upon planting that the seed labelled *P.*

heterophylla produced plants annual in habit; consequently the work on this species was restricted to inoculations made from a few mosaic *P. heterophylla* plants grown from roots brought in from the field.

Cross-inoculation from perennial species of Physalis to the cucumber. Since preliminary cross inoculation work indicated that the mosaic diseases of *P. pubescens* and *P. subglabrata* were identical, it was thought unnecessary to duplicate all the work done with *P. pubescens* on *P. subglabrata.* Since both *P. heterophylla* and *P. subglabrata* were susceptible to cucumber mosaic, it seemed probable that these perennial species were sources of primary mosaic infection to cucumber in the field. To prove that this was indeed the case, inoculations from mosaic *P. subglabrata* and *P. heterophylla* were made to cucumbers by means of aphids. The results of these experiments are given in table 8. Results of the reciprocal inoculations, mosaic cucumber to *P. subglabrata,* appear in table 9. These results show that the

TABLE 8.—*Results of inoculation of healthy cucumber plants by means of aphids from mosaic and healthy species of perennial Physalis*

Date inoculated	Source of aphids	No. aphids	No. plants	No. plants diseased	Date observed
March 29, 1924	Mosaic *P. subglabrata*	20	8	3	April 8, 1924
March 29, 1924	Healthy *P. subglabrata* (Control)	20	8	0	April 8, 1924
May 15, 1924	Mosaic *P. subglabrata*	20	8	2	May 23, 1924
May 15, 1924	Healthy *P. subglabrata* (Control)	20	8	0	May 23, 1924
March 22, 1924	Mosaic *P. heterophylla*	20	6	4	March 29, 1924
March 22, 1924	Healthy *P. heterophylla* (Control)	20	6	0	March 29, 1924
May 15, 1924	Mosaic *P. heterophylla*	20	4	1	May 23, 1924
May 15, 1924	Healthy *P. heterophylla* (Control)	20	4	0	May 23, 1924
June 27, 1924	Mosaic *P. heterophylla*	20	6	1	July 7, 1924
June 27, 1924	Healthy *P. heterophylla* (Control)	20	4	0	July 7, 1924

mosaic diseases of cucumbers and perennial species of *Physalis* are intertransmissible. This fact, coupled with the ability of aphids to transmit the disease, makes the relationship of perennial wild *Physalis* species to cucumber mosaic seem an important one.

TABLE 9.—*Results of inoculation of healthy plants of Physalis subglabrata by means of aphids from mosaic and healthy cucumber plants*

Date inoculated	Source of aphids	No. aphids	No. plants	No. plants diseased	Date observed
	Cucumber				
March 26, 1924	Mosaic	20	12	2	April 23, 1924
March 26, 1924	Healthy (Control)	20	10	0	April 3, 1924
April 8, 1924	Mosaic	25	10	4	April 20, 1924
April 8, 1924	Healthy (Control)	25	5	0	April 20, 1924
May 20, 1924	Mosaic	20	10	2	May 30, 1924

Importance of perennial Physalis species in the overwintering of cucumber mosaic. The probability of perennial species of *Physalis* being an important source of primary mosaic infection led to the eradication of these plants, together with others, in the experimental area at Rockland, Wisconsin, during 1923. Wherever wild *Physalis* plants were found, they were removed, the area being inspected throughout the summer at intervals of approximately two weeks. At the end of the summer of 1923, the amount of mosaic at Rockland had been greatly reduced as compared with that of the preceding season; and, as the mosaic of milkweeds and wild cucumbers had been practically exterminated during the preceding seasons, it seems probable that the reduction in the amount of the disease was due in great part to the removal of the ground cherry, the only remaining wild host of importance. Similar results were obtained in 1924 and will be published in a forthcoming paper by Doolittle and the writer.

The results of these eradication experiments, and the fact that cross-inoculations have shown that the perennial ground cherries carry cucumber mosaic over winter, indicate that these plants are important as sources of primary infection to the cucumber in the spring. This may easily be the case, for mosaic plants of *P. subglabrata* have been observed at Rockland, Wisconsin, early in May, a month before cucumbers appear above ground. Insects, particularly aphids, may transmit the disease from these plants directly to the cucumber, or they may infect the garden species of *Physalis*, from which the cucumbers may later be infected. It is possible that cucum-

bers in the field are infected by both of these methods. Flea beetles are commonly present on species of *Physalis*, and preliminary work by Gardner and Kendrick (11) indicates that these insects may transmit the mosaic disease. Aphids are probably of greatest importance, however, and when mosaic once appears in a cucumber field and these insects are abundant, the disease spreads very rapidly. In the fall, cucumber plants die before the wild ground cherries, and aphids or other insects moving to these plants which are still green may transmit cucumber mosaic to them, thus establishing a source of primary infection the following spring. The number of mosaic perennial *Physalis* plants is likely to increase constantly, since they are susceptible to mosaic diseases of several types, and the disease is probably often transmitted from one wild host to another. The possible importance of this genus is increased by the fact that Gray lists eight perennial species of *Physalis*, representatives of which are widely scattered over the entire United States.

SUMMARY

1. The mosaic disease of *Physalis pubescens* L. is readily transmitted to the cucumber by means of the cucumber aphis, and cucumber mosaic is also transmissible to *P. pubescens* by the same means. Infection can be secured between these hosts by artificial inoculation also, but the percentage of infection is lower by this method.

2. The mosaic diseases of pokeweed, tobacco, and tomato are also transmissible to *P. pubescens*.

3. *Physalis pubescens* is an annual, and the disease is not transmitted through the seed.

4. It has been found that a mosaic disease lives over winter in two perennial species of *Physalis*, *P. subglabrata* and *P. heterophylla*. The mosaic disease on these plants is readily communicated to the cucumber by means of the cucumber aphis and by artificial inoculation, and is also transmissible to *P. pubescens*.

5. Field observations indicate that mosaic infection is common on perennial species of *Physalis* in the vicinity of cucumber fields, and the results of field experiments indicate that these wild hosts are an important source of primary infection for the cucumber.

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DESCRIPTION OF PLATES

PLATE XXXI

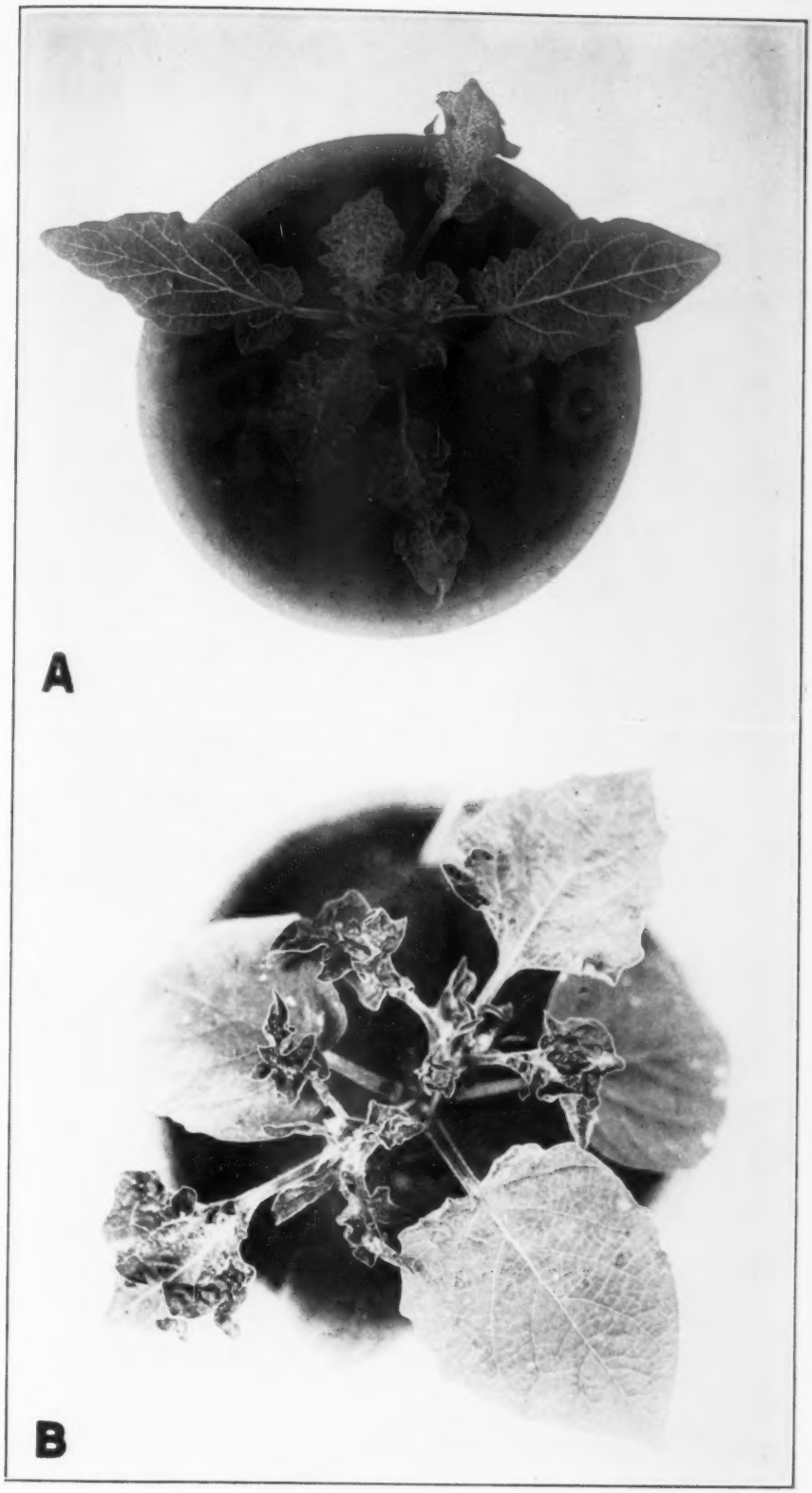
A. A mosaic plant of *Physalis pubescens* showing mottling, crinkling and dwarfing. Mottling of the type shown here is common in plants showing the first symptoms of the disease, the other symptoms appearing later.

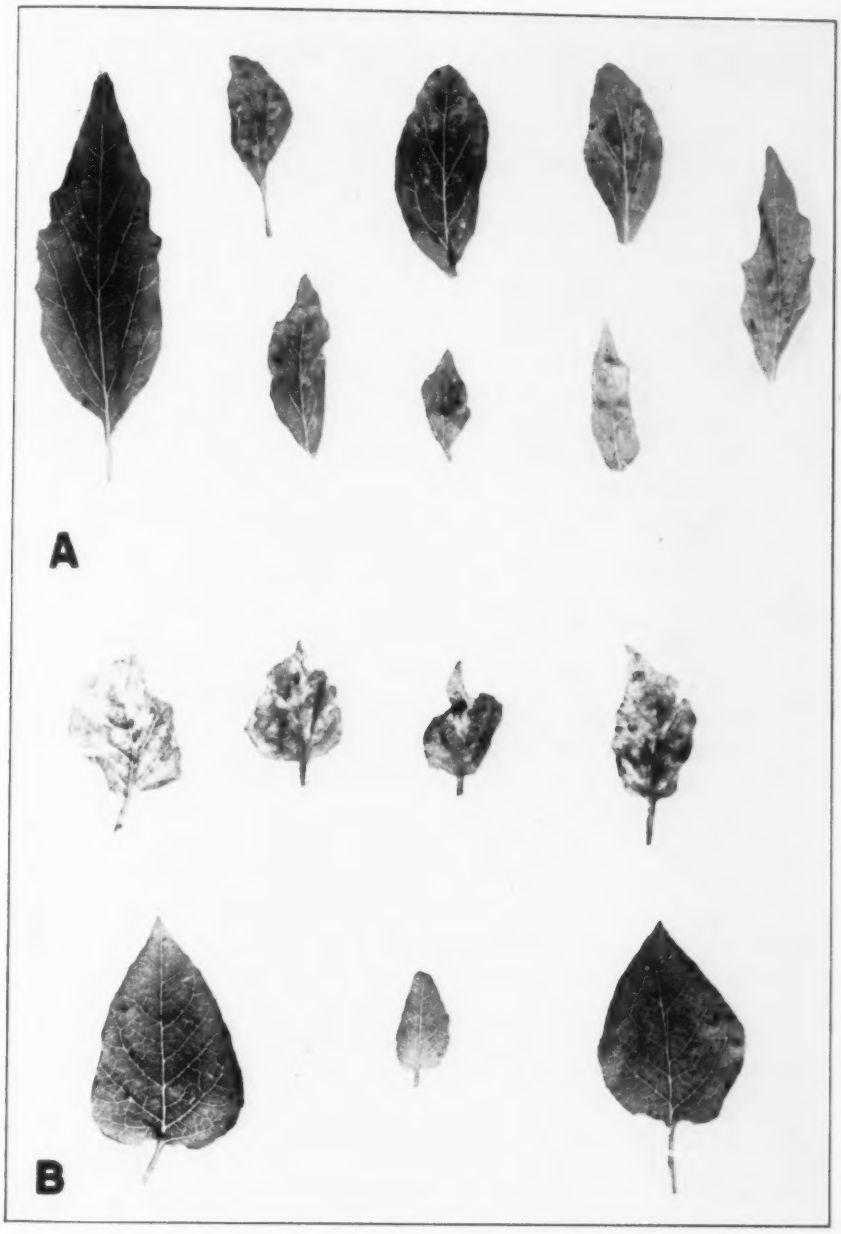
B. Mosaic plant of *P. pubescens* showing an acute degree of crinkling.

PLATE XXXII

A. Leaves of *Physalis subglabrata*; leaves at extreme right and left are healthy, leaves in center are mosaic.

B. Leaves of *Physalis heterophylla*; the upper row are mosaic, lower row healthy.


A
B


A
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ATTENUATION OF THE VIRUS OF SUGAR BEET CURLY-TOP

EUBANKS CARSNER[1]

WITH PLATES XXXIII TO XXXVII

STUDIES WITH CHENOPODIUM MURALE

The fact has been previously reported (2) that by passage of the virus of the curly-top disease of sugar beets through nettle-leafed goosefoot, *Chenopodium murale*, the virus is so attenuated that when transmitted to healthy young sugar beets it either fails to cause the curly-top disease or usually produces only mild cases. Thirty-three experiments have been recorded in which leaf-hoppers reared on *Chenopodium murale*, progeny of viruliferous parents, have been caged on one or more healthy sugar beets or other susceptible plants. These progenies, as demonstrated earlier (4), would under these circumstances have had to acquire the virus from the *C. murale* on which they were reared in order to transmit it to other plants. In twenty of the thirty-three tests, the plants to which the leafhoppers from *C. murale* were transferred remained healthy. In thirteen of the tests, some or all of the plants developed symptoms of curly-top. Nineteen of the sugar beets used in these thirteen tests developed the first symptom of the disease, the clearing of the small veins of the youngest leaves; and of these nineteen, fifteen subsequently lost all symptoms of the disease or developed only very mild cases, manifesting little or no dwarfing and only inconspicuous vein swellings.[2] In some of the tests, chickweed, *Stellaria media*, was used in addition to beet plants to test the leafhoppers from *C. murale*. Chickweed is very susceptible to curly-top infection. The details of one test with it are worth noting (Table 1).

The fact that the leafhoppers may secure the attenuated virus from *Chenopodium murale* does not interfere with their securing and transmitting the more active virus. Tests have been made in which leafhoppers reared on *C. murale* produced the mild form of curly-top in beets and were then caged for a few days on a plant affected with the severe form of the

[1] The writer wishes to acknowledge the collaboration of Mr. C. F. Stahl, formerly of the Office of Truck-Crop Insects, Bureau of Entomology, in some of the work reported, and the assistance of Mr. C. F. Lackey, junior pathologist, Office of Sugar-Plant Investigations, Bureau of Plant Industry, in some of the experiments.

[2] In some of the earlier of these tests records were made of the appearance of curly-top symptoms, but no subsequent record was taken. It is not, therefore, definitely known that four of the nineteen affected beets developed mild cases of curly-top.

TABLE 1.—*The attenuation of curly-top virus by Chenopodium murale, as indicated by inoculating Stellaria media with virus from Chenopodium murale*

Date	Results of inoculating *Stellaria media* with virus from *Chenopodium murale*	Results of inoculating *Stellaria media* with virus from a severely affected curly-top beet
Nov. 14, 1923	Seven leafhoppers reared on *C. murale* from viruliferous females were caged on *S. media*	Two leafhoppers from severely affected beet were caged on *S. media*
Nov. 19	Healthy	Diseased
Dec. 1	Diseased—faint but definite symptoms	Severely diseased
Dec. 4	No chlorosis	Marked chlorosis of terminal leaves
Dec. 17	Slight chlorosis of terminal leaves	Dead

disease and finally transferred to healthy beets. The beets to which these insects were at last transferred developed severe cases of curly-top. Beets affected with the mild form of the disease are neither rendered immune from nor more resistant to the severe form of the disease than are healthy beets. Leafhoppers from a severely affected curly-top beet have been caged on beets affected with the mild form of the disease, as well as on healthy controls. The symptoms of the severe disease developed in both lots.

STUDIES WITH RUMEX CRISPUS

Curly dock, *Rumex crispus*, is another plant which, with *Chenopodium murale*, was previously reported (1) as nonsusceptible to curly-top because nymphs reared on it from viruliferous females failed to produce curly-top when transferred to sugar beets. This result was obtained with an old dock plant which was transplanted to a pot from the field. Further study of the matter was undertaken when Dr. H. P. Severin stated to the writer that he had obtained curly-top symptoms on the dock by inoculating young plants. The later tests have been made with plants grown from seed. These tests show that *R. crispus* is resistant to the action of the virus and that the virus is attenuated by passing through it. It will make the results clearer to give the tests in detail (Tables 2 and 3).

STUDIES WITH SUAEDA MOQUINI

Another plant which is of interest in regard to attenuation of the curly-top virus is alkali blite, *Suaeda moquini* Greene. This perennial is probably

TABLE 2.—*Symptoms of curly-top produced on Rumex crispus inoculated by means of leafhoppers*

Exp. No.	Date	Treatment and results
1	Sept. 6, 1924	Two viruliferous leafhoppers caged on each of 12 *Rumex crispus* plants. Plants had 4 true leaves. Controls of same age were not inoculated. Insects removed Sept. 10.
	Sept. 17	Eleven of the 12 inoculated plants showed slight, irregular vein swellings on the youngest unfolded leaf.
	Oct. 11	The leaves of the diseased plants showed no marked evidence of dwarfing or rolling. Some of the leaves showed a few scattered, rough swellings of the veins on the lower surfaces, with distinct indentations of the upper surfaces over these swellings. The plants were evidently very mildly affected.
	Nov. 5	Definite symptoms of curly-top could be found only by very close examination of those plants which had previously shown clear-cut symptoms.
2	Sept. 3, 1924	On each of 4 young *R. crispus* plants, 6 viruliferous female leafhoppers were caged. On each of 4 similar plants, 6 nonviruliferous leafhoppers were caged.
	Nov. 5	One of the plants with viruliferous insects was definitely diseased, showing fairly conspicuous vein swellings and dwarfing.
	Nov. 18	The leaves of the plant affected on November 5 were about half as long as those of the controls. *R. crispus* was not nearly so severely affected as a beet of the same age would have been under the same conditions. The other three inoculated plants showed inconspicuous vein swellings and slight dwarfing (PLATE XXXIII). The weights in grams of the entire plants (leaves and roots) were: 4 controls, 31 to 38.5, total 142.5; 4 inoculated plants, 12 to 26, total 74.
3	Dec. 19, 1924	Seven viruliferous leafhoppers were caged on a *R. crispus* plant which had 5 true leaves. Seven nonviruliferous leafhoppers were caged on a similar plant. Insects removed after about 10 days.
	Jan. 19, 1925	A few very inconspicuous vein swellings could be found on the inoculated plant, but it was as large and as vigorous as the control.

TABLE 3.—*The attenuation of curly-top virus by Rumex crispus, as indicated by inoculating sugar beet plants with the virus from Rumex crispus*

Exp. No.	Date	Procedure and results
1	Nov. 17, 1924	From the 4 diseased *R. crispus* plants, marked A to D, of experiment 2, table 2, leafhopper nymphs were transferred to healthy sugar beets as follows: from A, 20 nymphs; from B, 15 nymphs to each of two beets; from C, 6 nymphs; from D 63 nymphs.
	Dec. 2	Beet A was too heavily loaded with insects—62 nymphs were recovered from it and transferred to a fresh healthy beet (AA).
	Dec. 3	Condition of beets: A was healthy; B′ and B″ showed conspicuous vein symptoms on the younger leaves—no evidence of dwarfing; C showed definite, conspicuous vein clearing of the three youngest leaves, but the symptoms were less advanced than in the B beets; D had not grown enough to show symptoms—discarded by mistake.
	Dec. 19	The four beets A, B′, B″, and C and beet AA all showed definite symptoms of curly-top—vein swellings—but all seemed only mildly affected.
	Jan. 21, 1925	All five of the beets had recuperated or continued as mildly affected cases. All showed vein swellings on the older leaves. Some showed faint clearing of the younger leaves, while with others the younger leaves appeared normal.
2	Nov. 6, 1924	Fifteen viruliferous adult leafhoppers were caged on a young *R. crispus* plant.
	Dec. 19	The *R. crispus* plant showed no dwarfing but did show scattered vein swellings on some of its leaves. Thirty-three nymphs were transferred from it to each of two healthy young sugar beets.
	Jan. 19, 1925	Both of the beets were healthy.
3	Dec. 23, 1924	Four leafhoppers reared on curly-top *R. crispus* (inoculation experiment No. 2) were caged on each of 20 small healthy sugar beet seedlings. On each of 20 similar seedlings 4 leafhoppers from a severely affected curly-top beet were caged.
	Dec. 31	Plants with insects from *R. crispus*: of 18 plants surviving, 7 were diseased. Plants with insects from curly-top beet: of 18 plants surviving, 18 were diseased.

TABLE 3.—(*Continued*)

Exp. No.	Date	Procedure and results
	Jan. 6, 1925	Plants with insects from *R. crispus*: of 18 plants surviving, 16 were diseased.
	Jan. 12	Contrast between the two lots of beets was beginning to give evidence of the attenuation of the virus by *R. crispus*. The first pair of true leaves of the plants infected with the virus from *R. crispus* were as curled and showed fully as conspicuous vein proliferations as did the corresponding leaves of the other lot, but were more nearly normal in length. The younger leaves (third and fourth true leaves) showed fewer conspicuous swellings on the veins and were noticeably less dwarfed than the corresponding leaves of the plants infected with the more active virus (PLATE XXXIV, A, B).
	Feb. 4	Contrast between the two lots was marked. All the plants infected with the attenuated virus were mildly and uniformly affected. Those of the other lot were severely affected (PLATE XXXIV, C, D).

of considerable importance in helping to tide the beet leafhopper over the dry periods of fall and early winter when favorable annuals are not yet available for food. Severin (3) reports collecting the insects from *Suaeda moquini* and from *S. depressa*, which closely resembles the species under consideration. The writer and his colleague, C. F. Stahl, have on several occasions collected *Eutettix tenella* from *S. moquini*. As opportunity has afforded, studies have been made of this plant as to susceptibility to curly-top and the effect of the plant on the virus. The tests thus far made may well be given in detail (Table 4).

ATTENUATION UNDER NATURAL CONDITIONS

The few experiments which have been made with *Suaeda moquini* clearly show, in the writer's opinion, that this species is resistant to the injurious effect of the curly-top virus and attenuates that virus in a way comparable with the two other species discussed. The attenuation which it causes probably indicates the explanation of the facts discovered in testing leafhoppers collected from the vicinity of Bakersfield, California. As will be explained further, a considerable proportion of these insects were found to have the curly-top virus in an attenuated condition. It is not meant that the *Suaeda* was responsible for all the attenuation which occurred, but that this attenua-

tion was brought about by the passage of the virus through uncongenial host plants.

TABLE 4.—*Attenuation of curly-top virus by Suaeda moquini, as indicated by inoculating sugar beet plants by means of viruliferous leafhoppers from Suaeda moquini*

Exp. No.	Date	Procedure and results
1	Feb. 21, 1924	Four adult leafhoppers collected from *S. moquini* near Bakersfield, California, were caged on a healthy sugar beet seedling.
	Mar. 5	Beet was healthy. Leafhoppers having the unattenuated virus would have produced curly-top symptoms in such a beet in less time than had elapsed.
	Mar. 18	The beet showed symptoms of the mild form of curly-top.
2	Sept. 18	On a small *S. moquini* plant, three viruliferous female leafhoppers were caged.
	Oct. 28	Leafhopper nymphs had hatched out on the plant. Five of these nymphs were transferred to each of five healthy young sugar beets.
	Nov. 4	One of the five beets showed faint but definite veinlet clearing, the early symptom of curly-top.
	Nov. 7	The veinlet clearing noted on November 4 had become very inconspicuous, but faint veinlet clearing was visible on the next smaller leaf, the youngest. Apparently the beet was but mildly affected.
	Dec. 3	The diseased beet showed vein swellings—very inconspicuously—on only one leaf, a leaf now mature. The plant was not dwarfed. The other four beets were healthy.
3	Nov. 7 and Nov. 17	On November 7, five more nymphs were transferred to each of two young healthy beets from the *Suaeda* plant of test No. 2; and on November 17, 25 nymphs were transferred to another beet.
	Dec. 19	All three beets were healthy. The *Suaeda* plant (inoculated September 18) had grown vigorously, and no evidence of the disease could be found on it.
4	Nov. 6, 1924	On each of four *S. moquini* seedlings, about three inches tall, five viruliferous male leafhoppers were caged. On each of two similar plants, five nonviruliferous males were caged.

TABLE 4.—(*Continued*)

Exp. No.	Date	Procedure and results
	Dec. 15	All four inoculated plants were stunted. The controls were approximately twice as large (PLATE XXXV, C, D). Close examination revealed occasional irregular swellings on some of the leaves of two of the affected plants (PLATE XXXV, A, B). These two plants had been kept against the south wall of the greenhouse—a position better lighted and relatively warmer than that where the other two inoculated plants were kept. The two latter plants did not show the leaf swellings. The viruliferous insects were removed, and ten nonviruliferous insects were caged on each of the four inoculated plants.
	Dec. 18	The leafhoppers were transferred from each *Suaeda* plant to a separate young healthy beet.
	Jan. 6, 1925	One of the beets showed the early stage of vein clearing on a part of the youngest leaf. The other three were healthy.
	Jan. 21	Another one of the beets was noted as diseased. The age of the leaves showing symptoms indicated that the first symptoms had appeared about a week earlier. Both of the diseased beets were mildly affected. Record was not made of which *Suaeda* plants the insects came from which produced the disease. Probably they were from those two which showed the leaf swellings.

The tests with the beet leafhoppers from the Bakersfield district were made on young sugar beets in a greenhouse at Riverside, California. The insects were collected at various times in the late winter and spring of 1924 from sugar beet fields and wild vegetation. A total of 801 leafhoppers were tested either singly or in small groups. A few of the tests given in detail will suffice to show the trend of the results (Table 5).

The fact that, in the tests with the leafhoppers from the Bakersfield district, most of the insects failed to produce curly-top symptoms within periods ten to twenty days longer than is required by leafhoppers with the more effective virus, and that most of the cases of the disease which they did produce were mild cases, justifies the conclusion that a considerable proportion of these leafhoppers had the virus in an attenuated form. This deduction is supported by field observations at Bakersfield. In the first place, the small amount of disease evident on March 14 in two beet fields where leafhoppers were found present early in February was contrary to what would

TABLE 5.—*The attenuation of curly-top virus under field conditions, as indicated by inoculating sugar beet plants in the greenhouse by means of leafhoppers collected from sugar beet fields and wild vegetation*

Exp. No.	Date	Procedure and results
1	Feb. 25, 1924	Fifty-seven adult leafhoppers collected from sugar beet fields and wild vegetation were caged on healthy young sugar beets as follows: 5 on each of 4 small plants and 37 on one larger plant.
	Mar. 5	One of the plants with 5 insects showed symptoms of curly-top. The other 4 were healthy.
	Mar. 29	The diseased plant was mildly affected. The plant with 37 insects showed mild symptoms on its youngest leaf. Under the same conditions leafhoppers with the unattenuated virus would have produced the severe form of curly-top in six to ten days.
2	April 15	On the plant of test No. 1, which showed symptoms on March 5 and from which the leafhoppers from Bakersfield had been removed, 15 nonviruliferous leafhoppers were caged. Ten nonviruliferous insects were caged on a severely affected curly-top beet.
	April 23	From the mildly affected beet, 6 leafhoppers were transferred to one young sugar beet and 7 to another. From the severely affected plant, 5 insects were transferred to a young sugar beet plant and 4 to another. These latter plants were for controls.
	May 2	The plant with 7 insects showed symptoms of curly-top. The one with 6 insects was healthy. Both controls were diseased.
	Sept.4	The plant with 6 insects had developed a mild case of curly-top. The date on which symptoms first appeared had not been recorded. Both plants were still very mildly affected.
3	March 25	The insects used were collected from sugar beet fields and from wild vegetation—*Erodium cicutarium* and *Atriplex semibaccata*. From beet fields: 162 insects were caged singly on healthy beet seedlings and 36 on a larger beet plant. From wild vegetation: 81 insects were caged in odd lots on 6 beets. As a control, one viruliferous leafhopper was caged on each of 4 beet seedlings.
	March 31	Three checks diseased. All other plants healthy.
	April 2	Plants with insects from beet fields: 6 of those with single insects and the one with 36 insects showed curly-top symptoms. Plants with insects from wild vegetation all healthy.

TABLE 5.—(*Continued*)

Exp. No.	Date	Procedure and results
	April 10	Three of 4 checks diseased. Thirty-two diseased of 162 plants on which insects from beet fields were caged. Six plants with insects from wild vegetation all healthy.
4	April 22	Seventy-five leafhoppers collected from beet fields on April 19 were caged singly on healthy young beets. Twelve viruliferous leafhoppers were caged singly on similar plants.
	May 3	One of the 75 beets was diseased. Eight of the 12 checks were diseased.
	May 10	Ten of the 75 beets were diseased. Nine of the 12 checks were diseased.
	May 28	Twenty-two of the 75 beets were diseased—all mild cases. The 9 diseased checks were all severely affected.

have been expected if the leafhoppers had had the more active virus. Of course the relatively low temperatures of the period retarded the development of the disease, but it is improbable that this one factor entirely accounted for the situation. In the second place, a considerable proportion of the plants which were diseased early in the season (April 8) were only mildly affected later in the spring (May 16). When the latter observation was made, these mildly affected plants showed prominent vein swellings on some of the older leaves while the newer leaves were nearly normal in appearance. Observations in a commercial sugar beet field at Riverside, California, indicate that in this district a considerable proportion of the numerous beet leafhoppers present had the curly-top virus in an attenuated form comparable to that found at Bakersfield. On May 29, 1924, approximately twenty-five per cent of the plants showed disease. Basing their decision on the size of the plants, the amount of disease, and the number of leafhoppers present, some sugar beet growers would have ploughed up the field. On July 14, the field presented a much better appearance than had been anticipated. Nearly all the plants in the field showed symptoms of curly-top, but a large proportion of them were merely mildly affected. The grower reported a profitable yield from the field.

Further study of the effect of the attenuated virus on the growth of sugar beets was conducted in the late summer and fall of 1924. The findings can be made clearer by giving the procedure (Table 6).

TABLE 6.—*Results of inoculating sugar beet plants with attenuated and unattenuated curly-top virus by means of leafhoppers*

Exp. No.	Date	Procedure and results	
		With attenuated virus	With unattenuated virus
1	July 3, 1924	Twelve nonviruliferous leafhoppers caged on a mildly diseased beet which was inoculated by a leafhopper from the Bakersfield district on April 22.	Twelve nonviruliferous leafhoppers caged on a severely diseased beet—one of the controls of the test of April 22.
	August 12	Fifteen leafhoppers, adults and nymphs, from the mildly affected beet were caged singly on small healthy beet seedlings.	Ten leafhoppers, mostly nymphs, were caged singly on similar beets which were growing under the same conditions.
	August 18	Two of the fifteen were diseased.	All ten were diseased.
	August 23	Twelve of the fifteen were diseased.	All ten were severely affected.
	Sept. 13	Two plants remained healthy. Two which once showed symptoms now appeared entirely normal. Ten of the mildly affected plants weighed 307.5 grams (PLATE XXXVI, B).	One of the check plants was dead. Nine check plants weighed 12.4 grams (PLATE XXXVI, A).
2	Sept. 23	Fifteen adult leafhoppers which had been reared on the mildly affected beet in experiment 1 were caged singly on young healthy beets. Similar plants were left uninoculated under similar conditions.	Fifteen adult leafhoppers from a severely affected curly-top beet were caged singly on young healthy beets.
	Oct. 9	Eleven diseased — all mild cases.	Twelve diseased—eleven severely affected.
	Nov. 11	The diseased plants were dwarfed in comparison with the plants uninoculated but were less affected than the diseased checks (PLATE XXXVII). Ten of the mildly affected plants weighed 155 grams. Fourteen healthy plants weighed 625 grams.	Twelve of the fifteen were severely and uniformly affected and weighed 58 grams.

DISCUSSION

Inasmuch as the attenuation of a virus causing plant disease has not previously been reported, so far as the writer is aware, and because the developments at different stages in the experiments are significant, the details of the principal tests which demonstrated the attenuation have been given.

In view of the evidence that the virus which produces the mild disease is the same as that which causes severe curly-top, except for the fact that it is attenuated, and the essential similarity of symptoms in the two cases, the writer deems it advisable to consider the mild disease as a mild form of curly-top rather than as a distinct virus disease.

The question may arise as to why there are such regional variations in curly-top epidemics as have been observed between central and southern California. Epidemics of the severe form of the disease have repeatedly occurred in the central part of the state, while in the southern part the mild form of the disease has been the dominant type—at least during the past two seasons. There is no marked difference in the climate of the two districts and the floras are similar. A difference between the two regions, which may be significant, is that sugar beets have been grown continuously and extensively for many years in central California, while in the area under consideration in the southern part of the state the crop has been grown only intermittently and in relatively small acreages. In view of these facts, it may be that the virulence of the virus has been maintained in the one case because beets were available each season, while, in the other, attenuation resulted because the virus was carried over mainly in wild plants which caused attenuation. Attenuation unquestionably occurs in regions subject to epidemics of the severe disease, but is usually unnoticed because the severe form of the disease predominates and even masks the mild form. It is easy to understand how the unattenuated virus may be overwintered in an area where it is abundant because, in addition to the attenuating plants, there are other agencies which are known to carry the virus over winter and which do not attenuate it. These are: diseased beets from the previous season, susceptible (non-attenuating) wild annuals, such as *Erodium cicutarium*, and the viruliferous beet-leafhoppers themselves.

The question as to whether the attenuated virus may again become virulent is still open. The evidence which thus far has been obtained is not conclusive. It may be said, however, that, in the writer's tests, virulence has not been restored by quickly passing the attenuated virus through sugar beets or even more susceptible plants.

SUMMARY

The three species, *Chenopodium murale*, *Rumex crispus*, and *Suaeda moquini*, which have been found very resistant to the virus of curly-top, have been studied in regard to their effect on the virus. It has been found that, on passage through these plants, the virus becomes so attenuated that it causes merely a mild form of curly-top when transferred to healthy beets or other susceptible plants.

The attenuated virus has been found to occur in beet-leafhoppers when collected from their natural breeding areas, and it has been assumed that the attenuation in these cases had resulted from the passage of the virus through resistant plants, of which the three species before mentioned are probably only representatives.

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EXPLANATION OF PLATES

PLATE XXXIII

A. A leaf of a *Rumex crispus* plant showing curly-top symptoms. Note the inconspicuous swellings on the veinlets. On September 3, 1924, six viruliferous leafhoppers were caged on the plant from which this leaf was taken. Photographed November 18, 1924. (See Table 2, Exp. 2.)

B. Healthy *R. crispus* leaf from a plant on which six nonviruliferous leafhoppers were caged as a check. (See Table 2, Exp. 2.)

PLATE XXXIV

A. Sugar beets on each of which four leafhoppers from a severely affected curly-top beet were caged on December 23, 1924. Photographed January 12, 1925. (See Table 3, Exp. 3.)

B. Sugar beets on each of which four leafhoppers which had obtained the curly-top virus from diseased *R. crispus* were caged December 23, 1924. Photographed Janu-

ary 12, 1925. Note that the younger leaves show conspicuous symptoms of curly-top but that they are not so dwarfed as are those in A.

C. The same pot of plants shown in A. Photographed February 4, 1925. Eighteen of the 20 plants inoculated survived; all were severely affected.

D. The same pot of plants shown in B. Photographed February 4, 1925. Eighteen of the 20 plants inoculated survived; 16 were mildly affected.

PLATE XXXV

A. Tip of main branch of a healthy *Suaeda moquini*. On November 6, 1924, five nonviruliferous leafhoppers were caged on the plant shown in C, from which this tip was taken. Photographed December 15, 1924. × 2 approx. (See Table 4, Exp. 4.)

B. Tip of main branch of diseased *S. moquini*. Note papillae on leaves. On November 6, 1924, five viruliferous leafhoppers were caged on the plant shown in D, from which this tip was taken. Photographed December 15, 1924. × 2 approx.

C. Control *S. moquini*. Five nonviruliferous leafhoppers were caged on this plant on November 6, 1924. Photographed December 15, 1924.

D. Curly-top of *S. moquini*. Five viruliferous leafhoppers were caged on this plant on November 6, 1924. Photographed December 15, 1924. Note size in comparison with healthy control. From *Suaeda* plants thus affected, the attenuated virus has been transferred to sugar beets, producing curly-top symptoms.

PLATE XXXVI

A. Sugar beets with severe form of curly-top. On August 12, 1924, a leafhopper from a severely affected curly-top beet was caged on each of these nine plants. Photographed September 13, 1924. Same magnification as B.

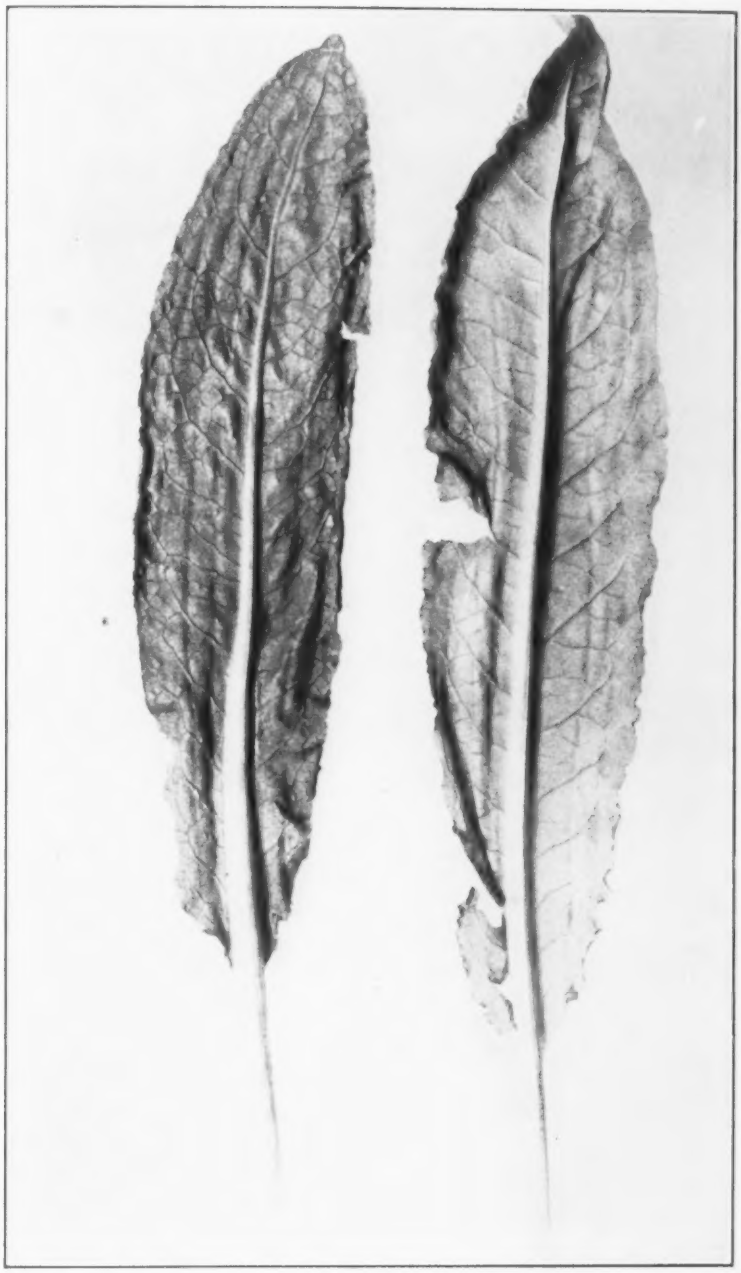
B. Sugar beets with mild form of curly-top produced by attenuated virus. On August 12, 1924, leafhoppers which had obtained the virus from a mildly affected beet (inoculated by a leafhopper from Bakersfield, Calif.) were caged on these ten plants, one insect to a plant. Photographed September 13, 1924. Note vein swellings (curly-top symptoms) on every plant, and compare size of plants with those in A.

PLATE XXXVII

A. Healthy sugar beet. Control for comparison with B and C.

B. A leafhopper with the virus from a severely affected curly-top beet was caged on this plant September 23, 1924. Photographed November 11, 1924.

C. Mildly affected curly-top sugar beet. A leafhopper with the attenuated virus was caged on this plant September 23, 1924. Photographed November 11, 1924.



A B

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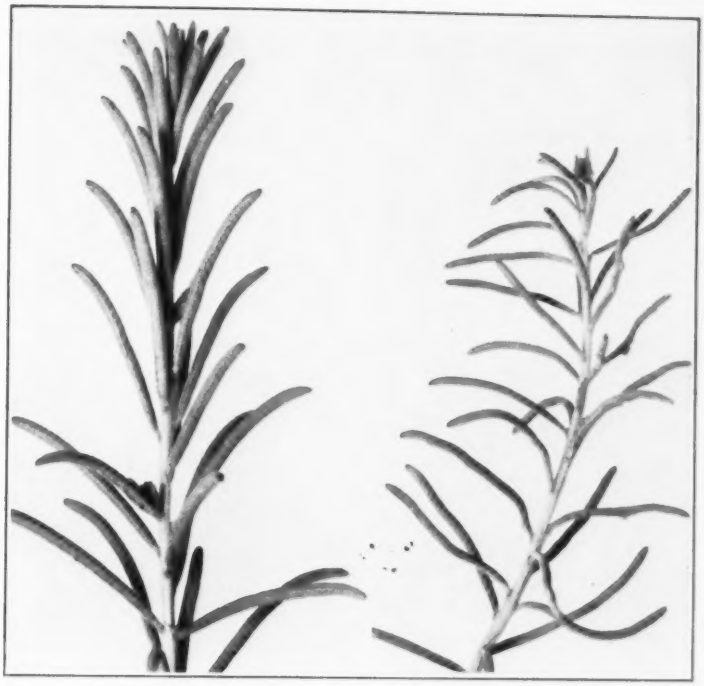

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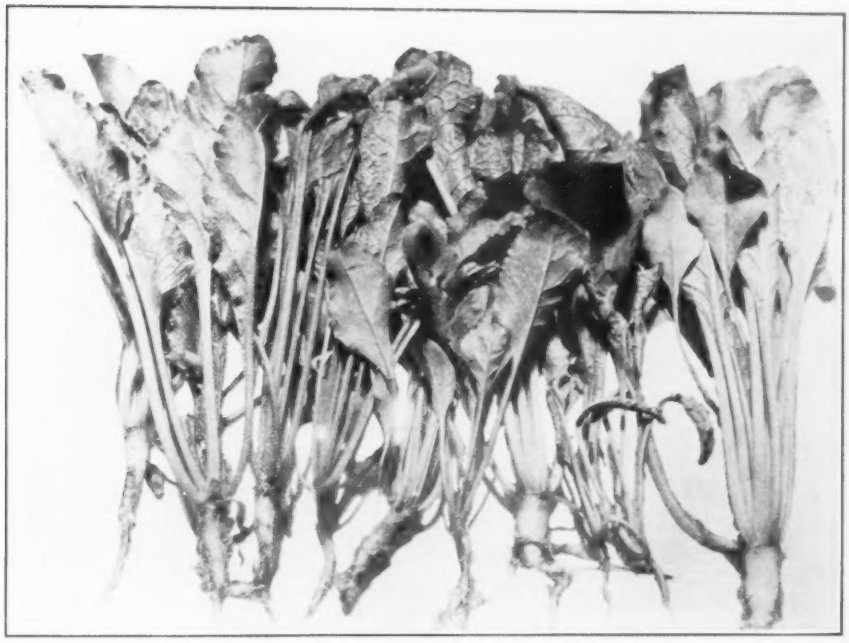

B

A
B
C

HYPERPLASTIC CRUSHING OF THE TRACHEAL TUBES IN MOSAIC TOMATO STEMS[1]

MAX W. GARDNER[2]

WITH PLATES XXXVIII AND XXXIX

In a previous paper (3) it has been shown that the necrotic regions in the fruits of tomato plants affected with the severe streak or winter blight type of mosaic are frequently accompanied by a hyperplastic or proliferative response. In a partial review of the literature it was shown that internal as well as external necrosis was a very common symptom of the mosaic types of disease, and that hypertrophy and hyperplasia were sometimes associated with this necrosis.

The necrotic surface streaks on mosaic tomato stems, as well as internal necrotic lesions, have been described by several observers, including, in addition to those mentioned in the paper to which reference has been made, Vanterpool (7) in Canada. Inasmuch as a certain type of necrotic streaking of the stem in greenhouse tomatoes seems to be a reliable indication of the presence of mosaic, it is of interest to note that there are references to this trouble which antedate Bailey's (1) account. Galloway (2, p. 347), in 1889, cites a letter from a grower in which the necrotic symptoms of mosaic are described, and an earlier and more unmistakable record is contained in a similar letter quoted by Plowright (6) in 1887.

A microscopic study of these mosaic lesions has shown that the necrosis may occur in any of the tissues of the stem and petiole, and that frequently there is a vigorous hyperplastic response. Under certain conditions this hyperplastic or proliferative growth is so directed as to crush the tracheal tubes. Unstained razor sections of fresh material, and microtome sections of preserved material stained with Delafield's haematoxylin, have been studied.

The necrosis may occur in close proximity to the growing point in the very young regions of the stem. Hypertrophy of the adjacent cells may be present in this very young tissue, but extensive hyperplasia is found usually somewhat further back from the growing tip. This would indicate that the cell proliferation follows the necrosis and is a direct response to it.

[1] Contribution from the Botanical Department of Purdue University Agricultural Experiment Station.

[2] The writer is indebted to Professor H. S. Jackson and Dr. E. J. Kraus for helpful suggestions and advice.

The necrotic pockets in the pith resemble those found by Kunkel in mosaic corn (4) and sugar cane (5). The affected cells collapse, and cavities of various shape and size are formed, each lined with the collapsed necrotic tissue. Hyperplasia of the adjacent pith cells occurs commonly, especially in very young pith (Pl. XXXIX, B) and when the necrosis is located near the outer regions of the pith. The outer regions of the pith contain the internal phloem strands, and the hyperplasia frequently originates near these strands in cases where there is no such response on the part of the pith cells lying on the inner side of the necrotic region. The hyperplasia in stems and petioles resembles that previously described in the fruit, and consists of zones, cushions, or whorls of thin-walled meristematic cells, devoid of intercellular spaces, growing in toward and crushing the necrotic cells.

Necrotic strips in the cortex, which constitute the externally visible brown streaks, may be internal or may extend to the exterior, and may result in surface grooves or crevices, owing to the collapse of the necrotic tissue. The necrosis may occur in the epidermis, the subepidermal cells, the collenchyma, or the cortical parenchyma. Necrotic zones in the inner portions of the cortex may be attended by hyperplasia in the shape of a cushion of thin-walled rectangular cells growing outward from the phloem region against the necrotic zone (Pl. XXXIX, D). This response was found most conspicuously developed in the base of the petiole. Necrotic surface streaks are frequently accompanied by hyperplasia of the underlying cortical cells, occasionally with the resultant formation of shallow surface blisters (Pl. XXXVIII, A) not unlike those previously described on the fruit.

The most vigorous hyperplastic response occurs, however, when the necrosis is in close proximity to the cambium, in which location plates of necrotic tissue, parallel to the cambial zone, seem to occur very frequently. These necrotic zones or planes are accompanied by a proliferative growth which apparently originates in the phloem region and pushes radially inward against the necrotic tissue and the xylem (Pl. XXXVIII, B). Under the hand lens, this growth is clearly visible as a glassy zone, measuring sometimes as much as 600 microns in radial thickness and bordered on the inner surface by a thin, brown, necrotic plane. The hyperplastic tissue is composed of short, thin-walled, closely packed cells quite unlike the xylem elements normally present in this region (Pl. XXXVIII, C; Pl. XXXIX, C). In longitudinal section, there occasionally has been observed an attempt at tracheal tube formation near the tangential edge of this hyperplastic tissue in the shape of an irregular row of short cells showing the characteristic wall thickenings.

These invasive hyperplastic growths are very similar to the internal intumescences found on the inner wall surfaces of the locules in mosaic

fruits, except that they are considerably elongated in a direction parallel to the axis of the stem. In some cases, however, rather wide arcs of the cambial circumference are involved. As in the case of the fruit intumescences, it appears that the hyperplasia in the stem tissues is a response to the necrosis and that its occurrence depends upon whether or not the necrosis is located in or near reactive tissue.

These internal growths develop considerable inwardly-directed pressure, because they not only flatten the necrotic cells but also invade the xylem and crush the tracheal tubes which happen to lie in their path (Pl. XXXVIII, C, D; Pl. XXXIX, A). The lumina of such tubes are completely obliterated, so that the latter obviously can no longer function as channels for the passage of water. In cases where a considerable proportion of the circumference of the xylem cylinder is invaded by these tube-crushing hyperplastic growths, it is readily conceivable that the water supply to the distal portions of the stem might thus be cut off partially, and it seems possible that this may account for certain effects of the disease, such as the wilting of young leaves.

SUMMARY

The severe streak or winter blight type of tomato mosaic is characterized by necrotic strips and pockets in all of the tissues of the stems and petioles.

This necrosis is frequently accompanied by hyperplasia or proliferation of the adjacent cells in the shape of zones or cushions of muriform tissue pushing in against the necrotic tissue.

When necrotic planes occur near and parallel to the cambial region, the inwardly-directed hyperplastic response is often so vigorous as to invade the xylem and flatten and crush the tracheal tubes.

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EXPLANATION OF PLATES

PLATE XXXVIII

A. Surface blister on mosaic tomato stem caused by hyperplasia of subepidermal cells underneath an area of necrotic epidermal cells. Longitudinal section, enlarged × 79.

B. Cross section of mosaic tomato stem showing necrotic planes in the xylem region pressed inward by the hyperplasia originating apparently in the phloem region. Enlarged × 30.

C. Central part of B enlarged × 74 to show the muriform, thin-walled tissue of the hyperplastic growth, and the flattening and crushing of the tracheal tubes upon which the abnormal growth impinges.

D. Cross section showing abnormal hyperplastic growth pushing inward against a flattened plane of necrotic tissue and crushing the tracheal tubes. Enlarged × 86.

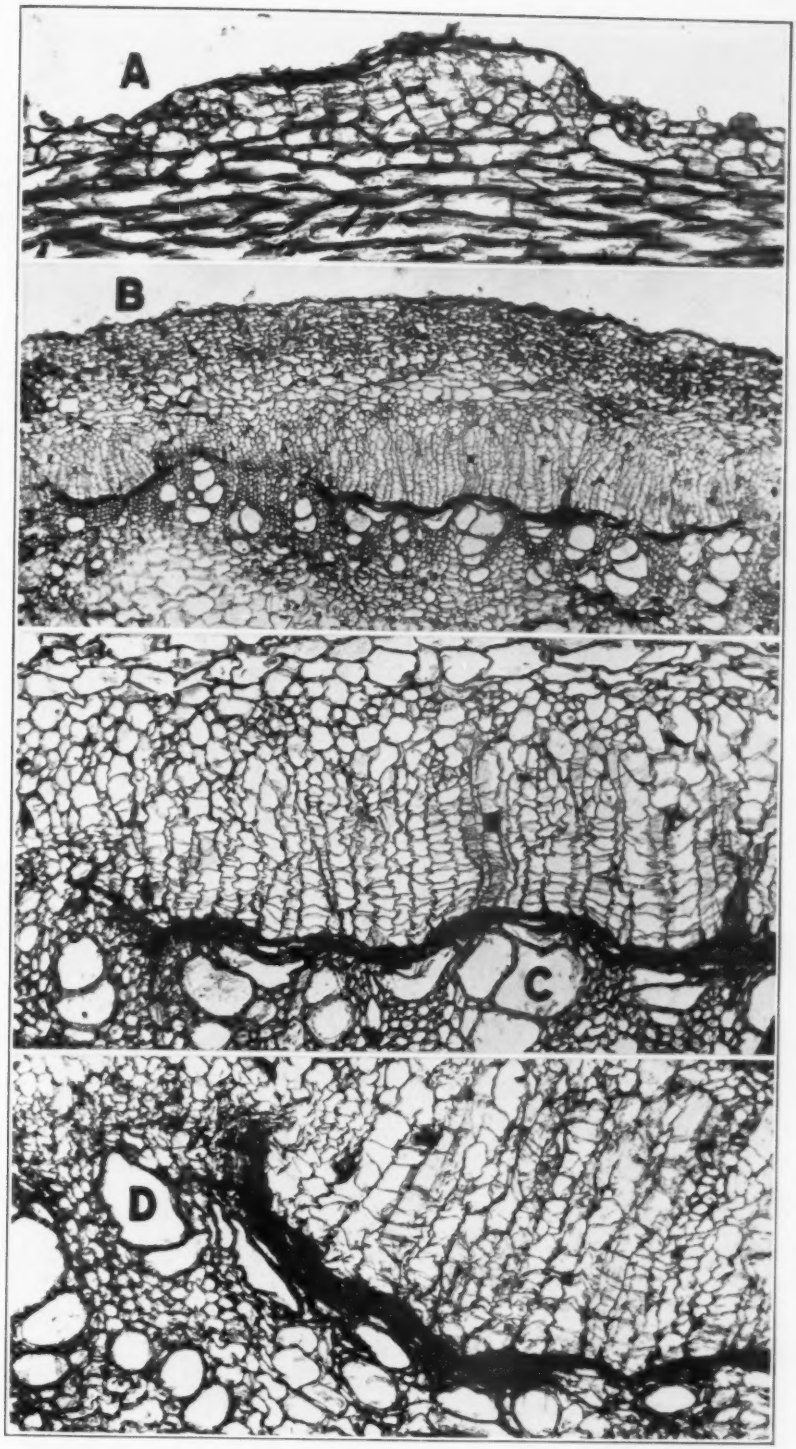
PLATE XXXIX

A. Cross section showing early stage of the crushing of the tracheal tubes by a hyperplastic growth. Groups of normal tracheal tubes are visible at the extreme right and left sides. Enlarged × 92.

B. Cross section of petiole showing necrotic zone in the pith accompanied by hyperplasia of the surrounding pith cells. Enlarged × 40.

C. Longitudinal section of the hyperplastic tissue pushing inward from the left against a necrotic plane and crushed tracheal tubes at the right. The cells are more or less rectangular and are not greatly elongated. Enlarged × 114.

D. Section through cortex near base of petiole showing a necrotic pocket in the cortex and a hyperplastic cushion pushing outward and crushing the necrotic tissue. Enlarged × 53.


A
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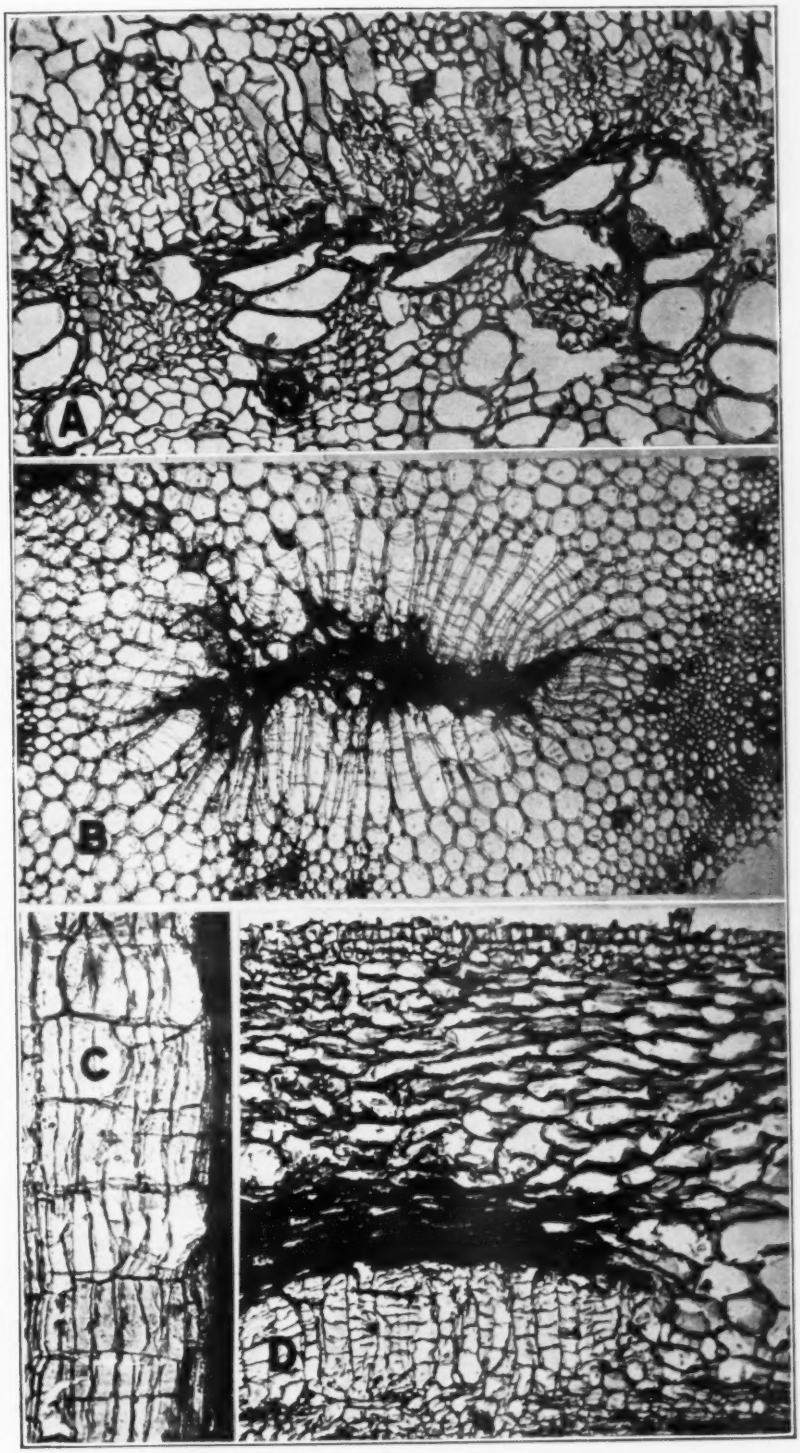
C
D


A
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C
D

THE MOSAIC DISEASE IN THE GARDEN PEA AND OTHER LEGUMES

S. P. Doolittle and F. R. Jones

With Plate XL

INTRODUCTION

Field observations of the past two years have shown that the mosaic disease of the garden pea (*Pisum sativum*) is widely distributed in Wisconsin. This disease was reported by Dickson (2) in 1922 as occurring in Quebec, and was also noted by Martin and Haenseler (5) in New Jersey in 1924. Mosaic plants were first observed at Madison, Wis., in 1923, and the disease was observed in many commercial fields in the state during 1924. Although it occurred in experimental plats at Madison in the latter part of June, the disease was not generally conspicuous in the field until about July 15. The pea disease survey (4) records it in 63 fields in various parts of Wisconsin. Many of these fields showed only a trace of the disease, but a considerable number contained from 5 to 15 per cent of mosaic plants. In general, although certain varieties seemed to suffer slightly from the disease, mosaic cannot be considered at present a serious menace to peas grown for the canning industry. A considerable amount of mosaic also occurred in 1924 on all varieties in the extensive pea trial grounds of Dr. W. B. Brotherton at MacMillan, Mich. Dr. Brotherton reports that late season varieties were severely damaged.

Among other legumes which show mosaic is the sweet pea (*Lathyrus odoratus*). Almost all of the sweet peas observed in the vicinity of Madison in 1923 and 1924 were infected with mosaic and severely damaged. The cultivation of this plant seems to have been generally abandoned in gardens at Madison because of this disease. Since the most common legume upon which mosaic occurs in gardens and near pea fields appears to be the red clover (*Trifolium pratense*)—a perennial upon which the disease overwinters—it seemed likely that the mosaic diseases upon these hosts might be intertransmissible. This paper is a record of studies of mosaic in these three species.

SYMPTOMS OF MOSAIC IN THE GARDEN PEA AND SWEET PEA

In the early study of mosaic in the garden pea, difficulty was sometimes experienced in distinguishing between certain distorted conditions of foliage occurring both in the greenhouse and field and true mosaic. Plants injured

by frost sometimes produce pale or crinkled foliage, suggesting a disease of the mosaic type. The mosaic studied here on the garden pea is characterized by a distinct mottling of the leaves, but there is little of the curling, wrinkling and general deformity of the leaf which occurs in the sweet pea and in mosaic plants of some other families. Mosaic pea leaves are usually a lighter green than those of normal plants and, in most cases, the mottled appearance is due to the presence of numerous small, dark green areas which occur between the larger veins (Pl. XL, fig. A). These dark areas are irregular in outline and usually seem to follow the small veinlets, but there appear to be none of the large green areas which occur in the case of such diseases as tobacco mosaic. Where plants have been mosaic for some time, the symptoms are often of a different type. In such cases, the dark green portions of the leaves may be replaced by yellowish-green areas which are of similar size and outline but which are lighter in color than the rest of the leaf.

In general, the leaves of mosaic peas are somewhat smaller than those of normal plants, and occasionally there is some slight curling of the edges of the younger leaves. Both symptoms appear most pronounced in the later varieties of more luxuriant growth. Such plants are occasionally considerably dwarfed by the disease, particularly if infected when small. In such cases, the pods appear to be smaller and fewer than those of healthy plants of the same varieties. The incubation period of the disease seems to vary from 6 to 14 days.

The mosaic disease of the sweet pea differs from the disease on the garden pea in that the plants are much more severely affected. The disease appears first in the younger leaves, and the symptoms at this stage are much like those of the garden pea. Occasionally, however, the contrast is greater between the dark areas and the remainder of the leaflet, which becomes a light greenish-yellow (Pl. XL, fig. C). As the disease progresses, the younger leaves develop a pronounced upward curling of the edges, which gives them a rolled appearance. Such leaves commonly develop small, elongated areas which are light yellow and appear to be thinner than the remainder of the leaf. These areas may be either raised or depressed, usually the latter, and appear much like the results of insect injury. Mosaic sweet peas are often much stunted in growth, not only of tops but of roots; and the plants sometimes appear to die of rootrot. The blossoms are streaked and paler than those of normal plants. A comparison made in 1924 showed that 32 mosaic plants produced only 85 blossoms in a period of 3 weeks from the time the first flowers appeared, while 38 healthy plants of the same varieties produced 187 blossoms in the same period.

INOCULATION EXPERIMENTS WITH MOSAIC OF THE GARDEN PEA

The inoculation experiments with the garden pea were conducted chiefly in the field, using the Alaska, Telephone, and Eclipse varieties. All of the plants inoculated were grown under cages covered with cheesecloth in order to prevent chance infection by insects. The inoculations were made by artificial methods and by means of aphids. In the artificial inoculations, the leaves and stems of mosaic pea plants were crushed in a sterilized mortar and used as inoculum. Inoculations were made from peas found infected in the field and also from plants experimentally infected from this source. The inoculations were made by pricking a few drops of the mosaic juices into 4 to 6 of the younger leaflets and by inserting a small fragment of crushed leaf tissue in a slight incision near the base and at the tip of the stem. The control plants were treated in the same manner, using the juices of healthy pea plants. All of the controls were in the same cages with the plants inoculated. In the aphis inoculations, pea aphids [*Illinoia* (*Macrosiphum*) *pisi* (Kalt.) Baker] were transferred from mosaic pea plants to the leaves of healthy peas under cages. Approximately 10 aphids were placed on each plant, and an equal number from healthy peas were transferred to each of the control plants, which were under a separate cage. These experiments are summarized in table 1.

TABLE 1.—*Results of inoculations to garden peas from peas affected with mosaic*

Date	Method of inoculation	No. plants inoculated	No. plants mosaic	Date observed
6/17/24	Artificial	13	5	6/30/24
do	Control	10	0	7/10/24
6/26/24	Artificial	27	8	7/ 8/24
do	Control	16	0	7/15/24
7/ 3/24	Aphids	30	23	7/18/24
do	Control	24	0	7/25/24
8/12/24	Artificial	20	10	8/30/24
do	Control	17	0	9/ 5/24
8/19/24	Aphids	16	16	8/29/24
do	Control	21	0	9/ 3/24

All of the varieties of peas used were susceptible to the disease, but the Telephone seemed more easily infected than either the Alaska or the Eclipse. In the inoculations with the Telephone pea, 70 per cent of the plants were infected, while with the Alaska and the Eclipse only 40 per cent of the plants became diseased. The transmission by aphids was also indicated in certain field plants which showed only a trace of mosaic until about 15

days after they became heavily infested by aphids, when a high percentage of the plants showed mosaic.

INOCULATION EXPERIMENTS WITH SWEET PEA MOSAIC

Taubenhaus (6), in 1914, reported the occurrence of sweet pea mosaic in Delaware and described a series of experiments which indicated that it was transmissible either by artificial inoculation or by aphids. A series of experiments by the writers has confirmed his results. These experiments consisted of artificial inoculations made in the same manner as described for the garden pea and also of experiments on the transmission of the disease by the pea aphis. The plants used in these experiments were grown in cages large enough to permit the worker to enter. Four varieties of sweet peas were used in the experiments: Glitters, Mrs. Cuthbertson, Flamingo, and Royal Scott, all of which proved susceptible to the disease. These experiments, as shown in table 2, have indicated that sweet-pea mosaic is transmissible either by aphids or by artificial inoculations.

TABLE 2.—*Results of inoculations to sweet peas from sweet peas affected with mosaic*

Date	Method of inoculation	No. plants inoculated	No. plants mosaic	Date observed
6/16/24	Aphids	63	50	6/28/24
do	Control	50	0	7/10/24
6/17/24	Artificial	20	12	6/28/24
do	Control	22	0	do
8/13/24	Artificial	10	6	8/25/24
do	Control	10	0	8/30/24

CROSS-INOCULATIONS FROM MOSAIC SWEET PEAS TO THE GARDEN PEA

Cross-inoculations, both by means of the pea aphis and by the use of the expressed juices of mosaic plants, have shown that sweet pea mosaic is transmissible to the garden pea. The Telephone and the Eclipse varieties were used under cages in the field. The results are given in table 3.

CROSS-INOCULATIONS FROM MOSAIC GARDEN PEAS TO THE SWEET PEA

Dickson (2) reported successful cross-inoculations from the garden pea to the sweet pea in 4 out of 23 plants, but did not draw definite conclusions from the experiment. The writers have found that the sweet pea is readily infected with the mosaic occurring on the garden pea. Thirty plants were inoculated by means of aphids taken from mosaic garden peas, 28 of which developed mosaic within 15 days. Aphids from healthy peas were placed

on 20 other plants as controls, all of which remained healthy. Artificial inoculations of 38 plants resulted in infection in 20 cases. The results of the above cross-inoculations have shown that the diseases of the garden pea and sweet pea are intertransmissible, and, since the symptoms produced on these hosts when inoculated from either the garden pea or sweet pea appeared to be the same, no distinction has so far been found between the diseases on these two plants.

TABLE 3.—*Results of cross-inoculations to garden peas from mosaic sweet-pea plants*

Date	Method of inoculation	No. plants inoculated	No. plants mosaic	Date observed
2/27/24	Aphids	23	11	3/14/24
do	Control	13	0	do
do	Artificial	19	0	3/19/24
do	Control	13	0	do
6/10/24	Aphids	32	22	6/21/24
do	Control	31	0	6/30/24
6/26/24	Aphids	31	16	7/ 8/24
do	Control	29	0	7/10/24
7/ 3/24	Artificial	16	8	7/15/24
do	Control	15	0	do
8/19/24	Aphids	37	35	8/30/24
do	Control	39	0	do
do	Artificial	18	10	do
do	Control	15	0	do

MOSAIC RED CLOVER AS A SOURCE OF INFECTION TO THE GARDEN PEA AND SWEET PEA

The frequent occurrence of mosaic on red clover has been mentioned previously. Davis (1) states that this perennial plant harbors the pea aphis during the winter and that the aphids migrate from the clover to pea fields in the early summer. If these migrating aphids can transmit mosaic from red clover to peas, the presence of mosaic in the pea fields thus can be readily accounted for. Field observations have indicated that the mosaic in red clover is transmissible to peas. In 1924, a number of rows of peas were planted parallel to a plat of red clover in which a large number of the plants were affected with mosaic. Observations made early in July showed that 96 per cent of the peas were affected with mosaic in the row adjoining the clover. In the next row the infection on the peas was somewhat less, and the reduction in the amount of mosaic continued in each succeeding row, the fourth showing only 50 per cent of mosaic. It seemed probable that the row adjoining the clover was more severely affected with mosaic

as a result of the greater ease and frequency of the aphis migration from the clover, but data of this sort are, of course, merely suggestive. Inoculations were made, therefore, to determine the susceptibility of the garden pea and the sweet pea to the mosaic on red clover.

CROSS-INOCULATIONS FROM MOSAIC RED CLOVER TO PEAS

Most of the inoculations from red clover to peas were made by the artificial method, using the same technique described in the case of other inoculations. The plants were grown in the field, and the controls were in the same cages with the inoculated plants. Two series of inoculations also were made with aphids. In this case the aphids from a cage of healthy peas were colonized on mosaic red clover plants in the greenhouse. After 24 hours the insects which remained on the clover were transferred to peas under a cage in the field. Aphids from healthy clover plants were placed on other pea plants as controls. The results of these experiments, as shown in table 4, indicate that the mosaic of red clover is transmissible to the garden pea either by means of insects or by artificial inoculation. It seems probable, therefore, that red clover acts as a source of mosaic infection to peas in the field.

TABLE 4.—*Results of inoculations to garden peas from mosaic red clover plants*

Date	Method of inoculation	No. plants inoculated	No. plants mosaic	Date observed
6/24/24	Artificial	18	6	7/ 8/24
do	Control	17	0	7/15/24
7/ 3/24	Artificial	17	8	7/14/24
do	Control	14	0	7/23/24
8/12/24	Aphids	10	7	8/22/24
do	Control	9	0	8/30/24
do	Artificial	24	12	8/23/24
do	Control	20	0	8/30/24
8/17/24	Aphids	8	6	8/28/24
do	Control	8	0	8/30/24
8/21/24	Artificial	27	11	9/ 6/24
do	Control	29	0	9/10/24
8/27/24	Artificial	23	8	do
do	Control	18	0	do

CROSS-INOCULATIONS FROM MOSAIC PEAS TO RED CLOVER

Some evidence has been secured in relation to the transmission of pea mosaic to the clover by aphids. Healthy red clover plants were grown in a cage with peas which were afterwards inoculated with pea mosaic.

Aphids were introduced into the cage after mosaic had developed on the peas, and a certain number of the insects migrated from the peas to the clover. At the end of 4 weeks, 6 out of 14 clover plants showed mosaic. Twelve healthy clover plants in another cage with healthy peas showed no signs of mosaic during the season, although aphids were present. Similar experiments are in progress.

CROSS-INOCULATIONS FROM MOSAIC RED CLOVER TO SWEET PEAS

As in the case of the garden pea, it has been found that the sweet pea is susceptible to the mosaic occurring on red clover. The inoculations in this case were made only by the artificial method and included 62 plants, 14 of which developed mosaic, while all of the 60 controls remained healthy. No inoculations have been made from the sweet pea to the red clover by the artificial method, but there is evidence that the pea aphis can transmit sweet-pea mosaic to this host. Healthy red-clover plants were grown in a cage of sweet peas which were inoculated with sweet-pea mosaic by means of aphids. After the aphids had been present for a number of weeks, it was found that 3 out of 10 of the clover plants had developed mosaic. Healthy clover plants in another cage of healthy sweet peas which were also infested with aphids showed no evidence of mosaic throughout the season.

TRIALS WITH SEED OF MOSAIC PEA PLANTS

Dickson (2) reported seed transmission of pea mosaic in certain varieties grown in the greenhouse, and, in a later abstract (3), mentioned further evidence of seed transmission in other varieties. In order to secure further evidence on this point, trials were made with seed collected by Dr. Brotherton at MacMillan, Mich., from 27 plants of the following varieties: Duchess of York, Prince Edward, Telephone, Carter's Daisy, Sharpe's Standard, and Eclipse. One hundred and sixty-two plants were grown in the greenhouse. The seed was planted on February 28, but no mosaic was noted on any of the plants until March 27, when they had reached a height of about 8 inches. On this date 7 plants showed signs of mosaic, but, since the disease had not appeared on the older leaves, it seems likely that infection came from other legumes in the houses, particularly since a few aphids had appeared in spite of efforts to control them by fumigation. During the spring of 1924, the remainder of this seed was grown under cheesecloth cages in the field. The seed was planted on June 15 and was covered with cages before the plants emerged. Four hundred and ninety-three plants were grown practically to maturity, none of which showed any evidence of mosaic.

In addition to this, another trial was made later in the season with seed collected from mosaic plants grown during 1924. The seed was collected from plants of the Alaska and the Ironclad field pea which developed practically all of this seed after their infection with mosaic. The seed was planted on August 16 under frames which were covered with cheesecloth. Due to the unusually cool moist summer, the plants made a vigorous and rapid growth and appeared to be little affected by the slight shading of the cloth. On September 11 the covering was removed and the plants examined for evidences of mosaic. At this time they had reached a height of 12 to 14 inches, but none of the 1,038 plants of Alaska and 388 plants of Ironclad showed any signs of mosaic. In order to make sure that the symptoms of mosaic had not been masked by the shading of the cheesecloth, the covers were not replaced after examination of the plants and they were left in the open until September 28 when they were again examined. On this date there was still no sign of mosaic infection. The experiments to date, therefore, have comprised 1,919 plants grown from seed from mosaic plants under insect-proof cages without the production of a single mosaic plant. These experiments are being continued on a larger scale in 1925.

CROSS-INOCULATION EXPERIMENTS WITH MOSAIC DISEASES OF OTHER LEGUMES

Cross-inoculations have been made from mosaic sweet clover (*Melilotus alba*) and bean (*Phaseolus vulgaris*) to garden peas and sweet peas without success. Further trials are in progress. The unsuccessful inoculations are summarized in table 5.

TABLE 5.—*Summary of unsuccessful cross-inoculations from mosaic sweet clover and bean to the garden pea and sweet pea*

Date	Plant used as inoculum	Plant inoculated	No. plants inoculated	No. plants mosaic	Date observed
2/27/24	Sweet clover	Garden pea	26	0	3/14/24
7/ 3/24	do	do	13	0	7/20/24
7/16/24	do	Sweet pea	22	0	8/ 7/24
8/12/24	do	Garden pea	16	0	9/17/24
8/21/24	do	do	19	0	do
do	do	Sweet pea	16	0	do
8/14/24	Bean	do	19	0	do
do	do	Garden pea	21	0	do
8/20/24	do	do	17	0	do

SUMMARY

1. The mosaic of the garden pea is widely distributed in Wisconsin, but it appears to cause little injury except in late season varieties. Sweet

peas, however, are severely damaged by mosaic. The symptoms of the two diseases are similar, but there is a greater mottling, dwarfing, and distortion of the leaves in the sweet pea.

2. The mosaic diseases of both the garden pea and sweet pea are transmissible by the pea aphis and by artificial inoculations. The mosaics occurring on these hosts are also intertransmissible by either method of inoculation.

3. Red-clover mosaic has been transmitted to the garden pea and sweet pea by artificial inoculations and to the garden pea by means of aphids. Inoculations by means of aphids also have indicated that red clover is susceptible to the mosaic occurring on these two hosts.

4. Since the red clover is a perennial on which the pea aphis is said to overwinter, it is probable that the aphids migrating from clover to peas in the spring act as carriers of the disease and thus introduce mosaic into the fields.

5. Over nineteen hundred plants have been grown from seed of mosaic garden and field peas, but there has been no evidence of seed transmission of the disease.

6. Cross-inoculations to garden peas and sweet peas from mosaic bean and sweet-clover plants have yielded only negative results.

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BUREAU OF PLANT INDUSTRY,
UNITED STATES DEPARTMENT OF AGRICULTURE.

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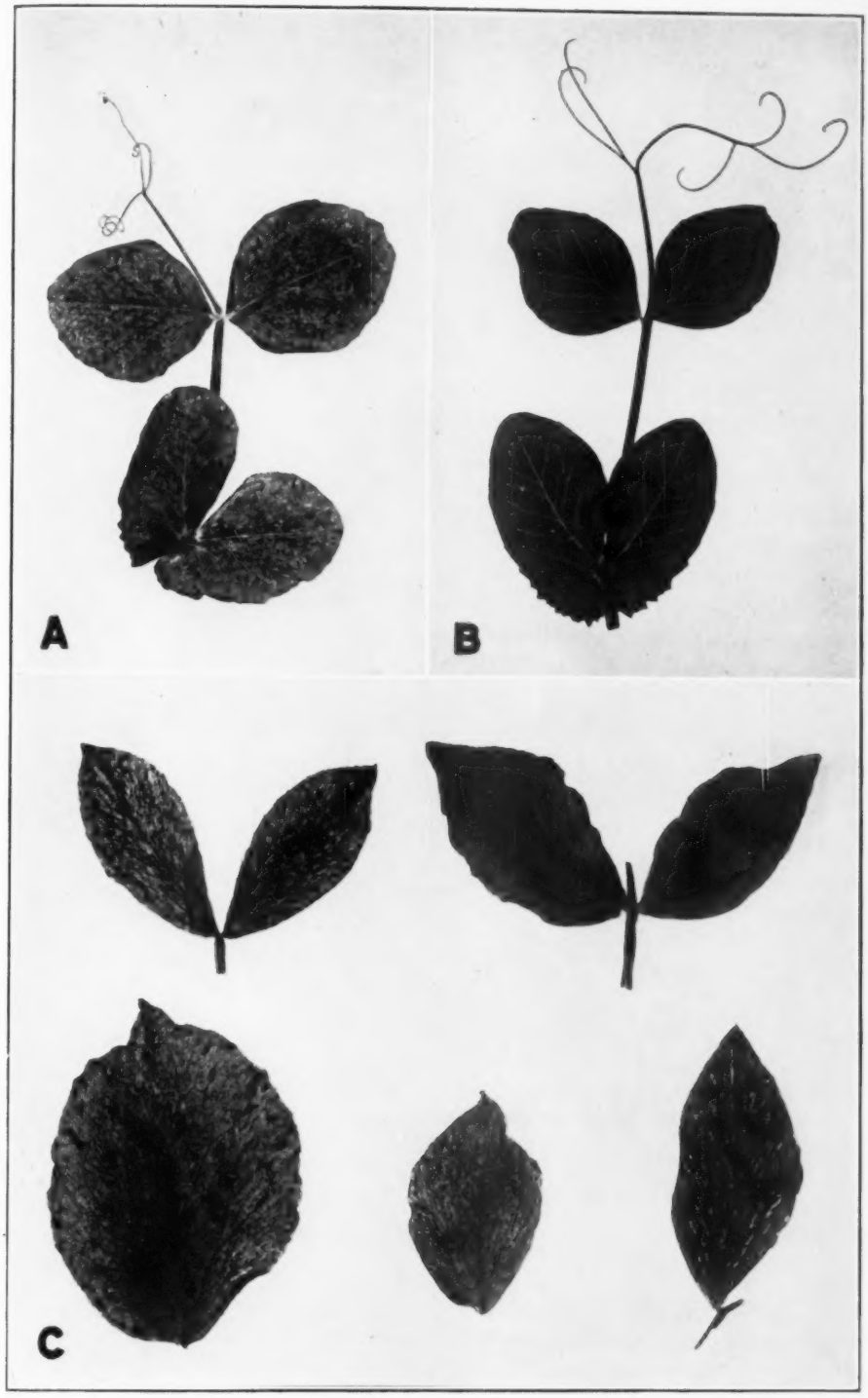
EXPLANATION OF PLATE XL

A. Mosaic pea leaf showing typical symptoms.

B. Healthy pea leaf.

C. Mosaic and healthy sweet pea leaflets showing various types of symptoms. Healthy leaflet in upper right corner.


A
B
C

ANTAGONISM OF THE WALNUTS (JUGLANS NIGRA L. AND J. CINEREA L.) IN CERTAIN PLANT ASSOCIATIONS

A. B. MASSEY

WITH FIVE FIGURES IN THE TEXT

The writer's attention has frequently been directed to the poor development and dying of tomatoes and other plants in the immediate vicinity of walnut trees. This is especially true of the black walnut, *Juglans nigra*, which is found in more frequent associations with crop plants than the butternut, *Juglans cinerea*. Review of the literature on the subject has not revealed any report of a definite study, but there are several notes which bear directly on the matter. Jones and Morse (2), in a study of the shrubby cinquefoil, *Potentilla fruticosa* L., a pasture pest in Vermont, record observations made by A. H. Gilbert on the effect of butternut on the cinquefoil. It was observed that around butternut trees there was a circular area, greater than the spread of the limbs, in which the cinquefoil was dead. At the borders of the area, dying plants were found. The death of the cinquefoil always indicated that the trouble had its origin in the roots. Examination of the root systems of dead and dying plants revealed in every case a close association between the roots of the weed and those of the butternut. In situations where rock outcrop interfered with the spread of the butternut roots, the cinquefoil grew normally.

> "Moreover, with such butternuts, the 'dead line' for the weeds is pushed outward year by year as the tree enlarges, so that the trees may be surrounded by a circle of dead and dying cinquefoil plants bordering the clean grassy plot under the tree. This antagonism is, we believe, attributable rather to the root relations of the two plants, than to those of shade. Thus young butternuts from 2 to 8 feet high were observed to be surrounded by a circle which might be twice the diameter of the top of the tree, within which the weeds were dead, with dying plants bordering its margins. Such butternuts do not cause much shade. Moreover, young birch, beach, maple, cherry, apple and pine trees in the same field showed no such striking relation to the death of the cinquefoil, healthy plants of the weed frequently growing close under their branches." (2, p. 189.)

In a short note, Mel. T. Cook (1) mentions having observed several cases of injury to tomatoes and potatoes in the near vicinity of black walnut trees. He writes:

> "Attention has been called from time to time to a number of cases of wilting of potato and tomato plants which was undoubtedly due to walnut (*Juglans nigra*) trees growing in the immediate vicinity. The plants show a pronounced wilting but

do not lose their color or die, as in the case of plants that have been attacked by wilt-producing fungi or bacteria, or struck by lightning. The range of the wilting coincides very closely with the spread of the root system. The plants may wilt uniformly within a large circle or there may be areas of wilted and areas of erect plants, which coincides with the distribution of the root system. In some cases the wilting was in a circular area around the trees in the field, in other cases in a semi-circular area next to trees along the margins of the fields. In all cases observed, the plants beyond the spread of the roots of the trees were normal. A number of cases have been investigated by the writer and there is no doubt as to the cause of the wilting. So far as the writer has observed, other crops are not affected by the walnut trees and other trees do not cause a wilting of crops or wilt vegetation."

Dr. A. W. Drinkard, Jr., Director of the Virginia Agricultural Experiment Station, has described to the writer orally the severe injury to experimental plats of tomatoes which, by chance, were planted near some black walnut trees on the edge of a field. When about half grown, the tomato plants wilted and died over a semi-circular area extending 30 to 40 feet from each walnut tree as a center. On one side of the plats was a small stream which kept the soil not only well supplied with water but was inclined to keep it too wet. It is evident from this observation that the effect of the walnut is not a case of water relation as might be suggested.

Fromme, in an unpublished note, describes disastrous results to tomatoes in Virginia:

"On several occasions in Virginia a wilting of tomato plants growing in proximity to black walnut (*Juglans nigra*) has been noted. The first observations were made near Amsterdam in 1916. Areas of wilted plants were seen in two separate fields of tomatoes, and each area centered on a walnut tree growing in the fence row. The rapid wilting of the plants suggested bacterial wilt (*B. solanacearum*), but examination failed to reveal the presence of this organism. On inquiry it was learned that the occurrence of this type of wilt in proximity to walnut trees was a matter of common observation among farmers in the locality.

"An opportunity for additional observations came during the season of 1917 in connection with a spraying experiment at Blacksburg. A number of small plots were provided in this test, each consisting of 16 plants set in the form of a square. The spacing between plants was 5 feet, with 7 foot alleyways between plats. The arrangement is shown in figure 1, together with the location of two black walnut trees, on the border of the field, which were overlooked at the beginning of the work. The varieties were Stone and Greater Baltimore. The date of transplanting was May 22; and the first wilting was noted approximately two months later, on July 26. The number of wilted plants increased rapidly as the season advanced, and by harvest all plants within the zone of influence of the trees showed marked injury. The majority of the plants were dead at this time, and fruit production throughout the areas affected was practically nil. The location of affected plants as determined at harvest time is shown in the diagram by means of the blackened squares. It will be noted that all plants within a radius of 50 feet of one tree and of 40 feet of the other were affected. There were 42 affected plants in the first area and 20 in the second.

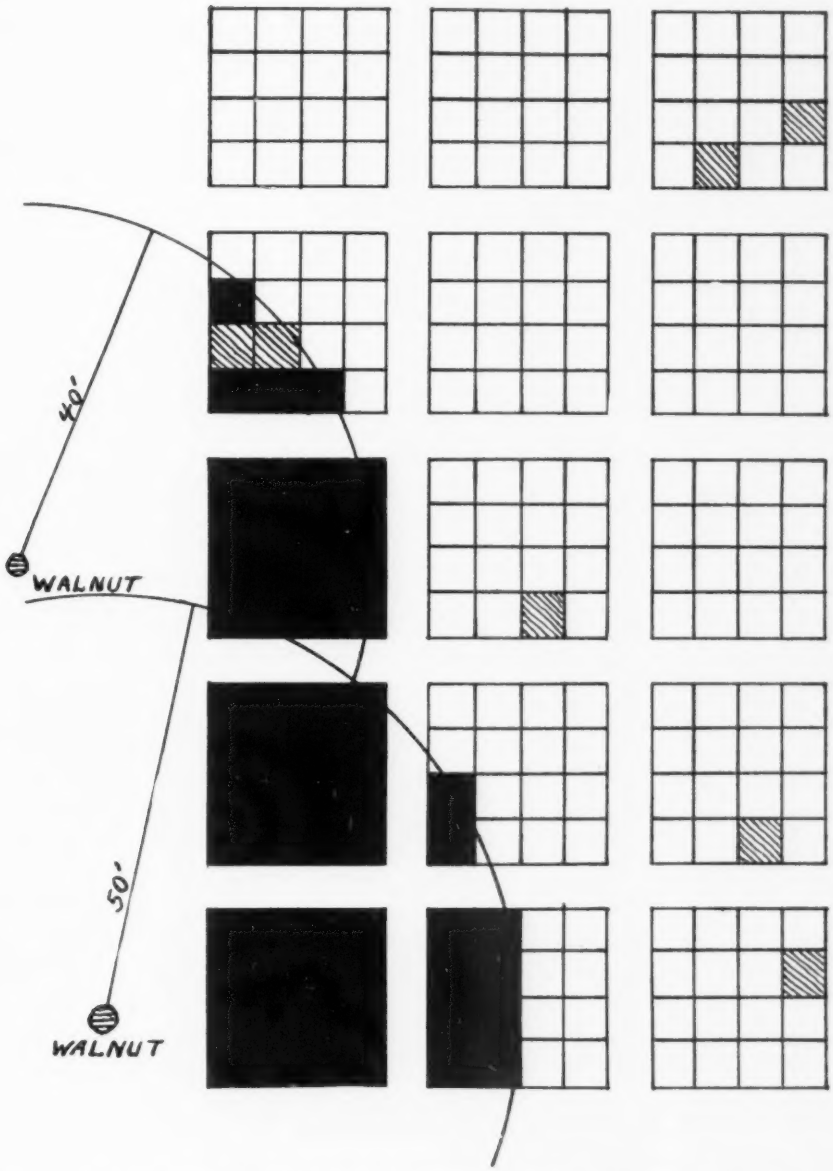


FIG. 1. Map showing location of wilted tomato plants with reference to black walnut trees. Black squares show wilted plants, white squares healthy plants, shaded squares missing plants. (Fromme.)

"The writer has made no study of the cause of this wilting, but the evidence suggests the effect of a toxic excretion from the walnut roots rather than a depletion of soil moisture. As Cook has noted, plants other than tomato and potato are not affected, and trees other than walnut apparently do not cause a similar effect."

The majority of the observations the writer is able to find relate to the effect of the walnut on tomatoes and potatoes. These plants seem to be especially susceptible. In an unpublished report for 1922 of the field laboratory for fruit disease research, located at Winchester, Va., F. J. Schneiderhan reports a case of injury to four apple trees equi-distant on as many sides of a black walnut tree. His note is as follows:

"A noteworthy case of incompatibility between trees of different species was observed in the experimental plats of the Stonewall Orchard. The first apple tree in Plat 1 of our scab experimental work was found to have dead branches on its west side. No cankers or other causes were found on the tree itself. A large black walnut tree stands 50 feet west of this apple tree. It was thought possible that a toxic influence of the *Juglans* species might have some connection with the killing of the branches mentioned. Upon investigation, it was discovered that other apple trees planted north, east and south of this same walnut tree were affected to a greater extent than the tree in our experimental plat. The tree located south of the walnut tree was a dwarf, the one on the north had not only lost all of its branches on the side facing the walnut tree but was also severely dwarfed, while the apple tree on the east was completely killed. From this we concluded that black walnut trees planted within 50 feet of apple trees exert a toxic influence which results in severe dwarfing and ultimate death of the latter."

Unverified reports of similar cases of injury to apple trees in other orchards in Virginia have been brought to the writer's attention.

Schreiner and Reed (3) state, "It is known that certain crops do not thrive when planted on newly cleared land which was originally covered with walnut or butternut trees." It is evident from these few authentic observations which the writer has been able to locate that there is a decided antagonism of species of *Juglans* to plants of different types. However, no one seems to have made a close study of the matter to determine the cause.

Antagonism in other plant associations has been noted by Schreiner and Reed (3), and by Schreiner and Skinner (5). These investigations will not be discussed here.

With the evidence in mind of an antagonism between the roots of *J. cinerea*, *J. nigra*, and certain weed and crop plants within their reach, the writer in 1923 decided to investigate the matter in the hope of obtaining some data as to the cause of the antagonism. Several possible explanations came to mind when planning the approach to the problem.

1. Is there an exhaustion of the soil water by the walnut trees, thereby causing wilting and death of the plants affected?

2. Is there a toxic substance developed in the soil from the decay of walnut leaves or nut shells around the tree?

3. Do the roots secrete a toxic substance which is detrimental to some plants?

The first and second questions were readily disposed of after careful consideration of the authentic cases described above. The condition of the soil in the tomato plats of Drinkard's experiments shows that it is not a

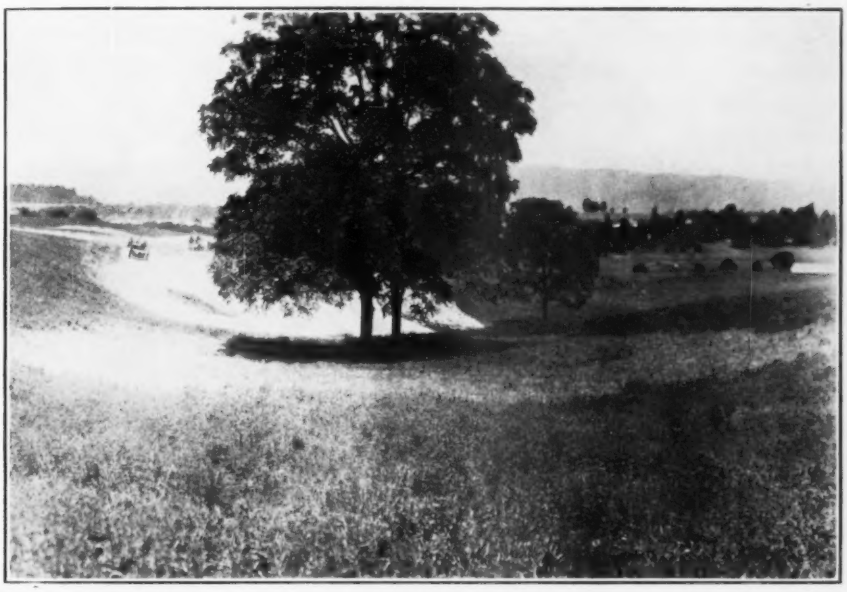


FIG. 2. Walnut trees in an alfalfa field, showing area in which alfalfa has been killed.

question of soil water supply, as this soil had an abundance of water. The unfavorable action of planting tomatoes on new ground which has recently supported growth of butternuts or walnuts also disposes of the first question, as the action on the plants occurs although the trees have been removed and could cause no depletion of the soil water. The situations of several trees closely observed by the writer disproved the probability of there being injurious substances formed through the decay of leaves and nut shells. These trees were so situated that strong winds prevented the accumulation of leaves and the accumulation of nut shells was not sufficient to cause any trouble. Were the second question to be answered in the affirmative, one would expect a more general and even dying of the plants over the area,

which is not always the case as will be shown later. The third explanation held the attention of the writer throughout the investigation. Do the roots of species of *Juglans* secrete a toxic substance which is injurious to some plants within their reach? To study this question, some walnut associations were examined critically and several experiments carried out.

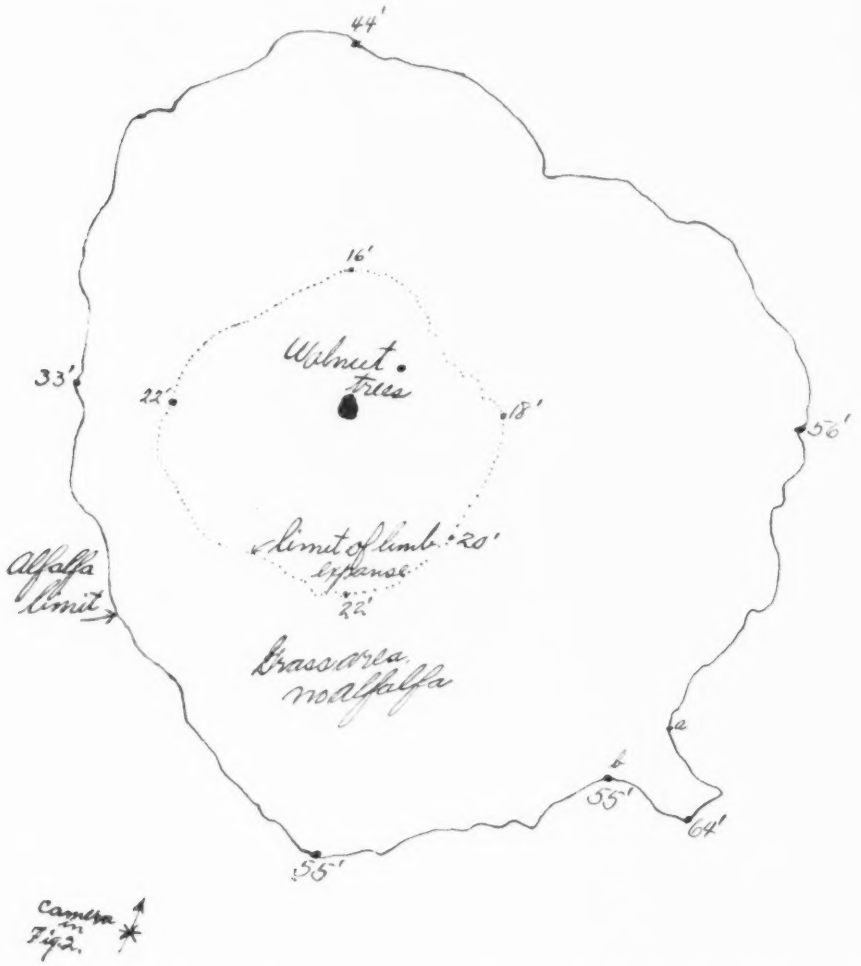


FIG. 3. Diagram of walnut trees shown in figure 2, indicating the position of the trees, limb expanse and area in which the alfalfa has been killed. Outside of area bound by the solid irregular line the alfalfa was green and healthy. Figures indicate distance from trunk of larger tree, given in feet.

WALNUT-ALFALFA ASSOCIATION

In a large alfalfa field of the Virginia Polytechnic Institute farm, there are two black walnut trees. One of these trees is of considerable size; the second is half dead through the work of borers. Around these trees it was observed that the alfalfa had been completely replaced by grass, as indicated in figure 2. The light area is entirely free from alfalfa and consists largely of timothy. Figure 3 is a diagram of the healthy tree and the border of living alfalfa plants. The limit of the limb expanse is shown by the dotted line, and the limit of the alfalfa-free area is shown by the irregular solid line. The extent of this area at first seemed to be too great in some directions to be equal to the expanse of the roots under ground; however, upon close examination of the first foot of soil at various intervals, it was found that walnut roots in every case reached to the extreme edge of the alfalfa-free area. At one edge of this area there was a marked extension, the end of which was 64 feet from the base of the walnut tree and 9 feet from the general border of the alfalfa-free area. This area was dug carefully to find whether walnut roots could be found within it. A trench was dug from point a to b, and a living walnut root three-fourths of an inch in diameter was found. The continuation of this root was such as to cause the irregular shape of this little area. The offshoot to the left was found to coincide with the development of a small end of the root underground. In other words, as the root came in contact with the alfalfa roots, the latter were killed. This case shows very strikingly that walnut trees are very injurious to alfalfa and that the dying of the plants is very closely associated with the development of the roots of the tree. There seems to be no general diffusion of any injurious substances through the soil; the action is always in the immediate vicinity of a walnut root. From this diagram one can also see that the dying of alfalfa could not be caused by shade; neither could it be caused by the decay of leaves, since the area is too large for the leaves of the trees to cover sufficiently to cause injurious action.

ASSOCIATION OF WALNUT WITH CERTAIN PLANTS IN HOME GARDENS

Four rows of tomatoes were planted specifically to study the action of the walnut on tomato plants. In the center of a general garden, four rows of tomatoes were planted in the vicinity of a walnut tree, as shown in figure 4. The tomatoes were set out on May 20. In the latter part of June, plants began to wilt and die. The wilting of the tomato plants was uneven over the area, and often one or two branches on the side of a plant became permanently wilted while the rest of the plant was healthy. Upon carefully removing the soil to expose the situation of the roots, it was found

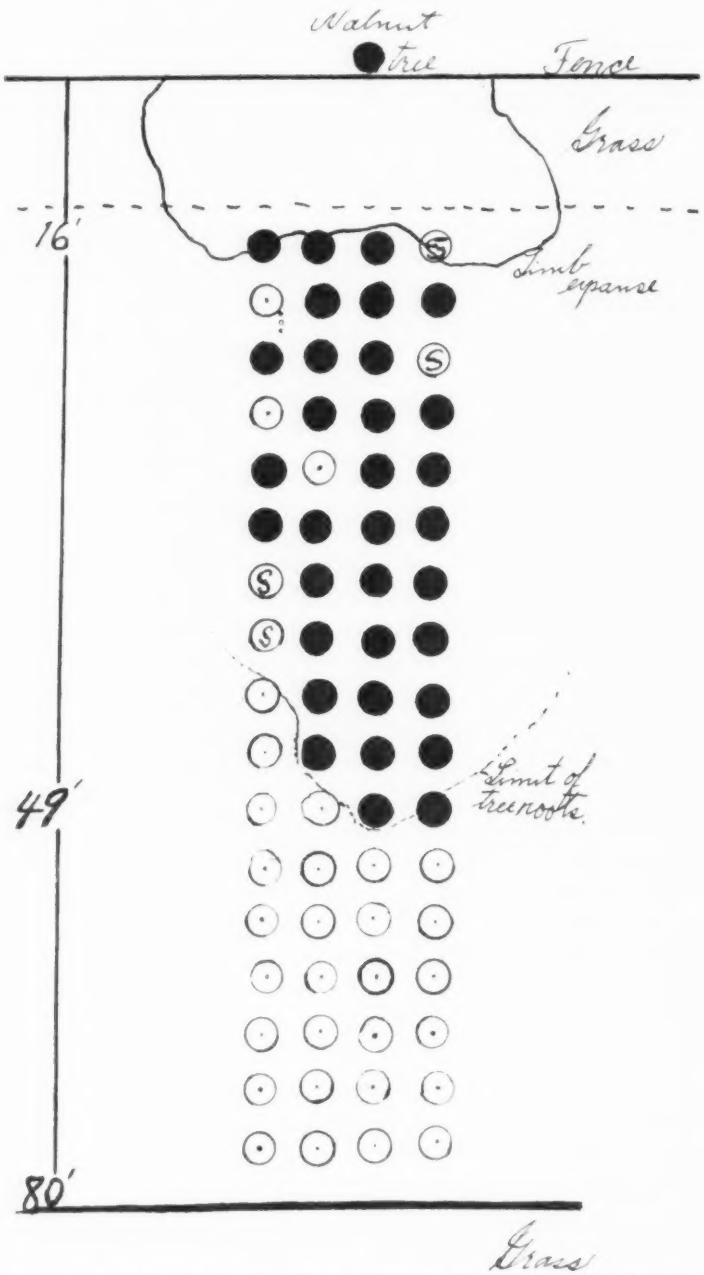


FIG. 4. Diagram showing condition of tomato plat eight weeks after setting plants in the immediate vicinity of a black walnut tree. Each circle indicates position in which a plant was set. White circles indicate plants which remained healthy. Circles with S in them represent plants that died soon after transplanting. Black circles indicate plants that wilted and died.

that in every case there was close contact between the tomato roots and those of the walnut. The plants wilted in a line sometimes diagonally across a patch, and sometimes parallel with the rows of tomatoes. This, it was found, was governed by the position of the walnut roots. In cases where one part of the plant wilted and the rest of the plant remained healthy, it was found that a walnut root ran between the rows or to one side of the plant and that one root came in close contact with those of the walnut, this one root always arising from the side of the plant on which the wilted branch was located. When the other portion of the plant began to wilt, it was found that roots from that side of the plant also had come in close touch with walnut roots. The direction of the roots underground could be traced without removing any dirt, by observing the development of wilt in the tomato plants. The probable distribution of the roots was predicted and later verified by removal of the soil. The presence of three unaffected plants within the affected area, marked by clear circles in figure 4, was at first not understood. They were well within the affected area and showed no signs of wilting. However, after digging them it was found that the roots of these plants did not come in contact with the roots of the walnut. The old walnut roots seemed to have as much effect on wilting of the plants as the young, actively growing ones. There was no specific relationship between the region of the strongest activity of the walnut roots and the wilting of the tomatoes, as would be expected if the trouble was due to lowering of the soil moisture.

On the right of the tomato patch, diagrammed in figure 4, irish potatoes were growing. There was a marked effect of the walnut on these potato plants, although it was not so distinct as on the tomatoes. Within the walnut-root invaded area of the garden there were also growing, in good condition, beets, snap beans, and corn, none of which showed any signs of wilting.

To determine whether the action on the tomatoes was one of toxicity from the walnut root, several pieces of bark from walnut root were placed in a water culture of tomato plant (fig. 5). Within 48 hours, plants in the water culture containing pieces of the bark were wilted and their roots browned, while the check plants in a similar solution with no walnut bark were erect and in good condition, thus indicating that there is some substance in the bark which is detrimental to the development of the tomato. In another experiment, large tubs of more than a bushel capacity were partly filled with soil and several pieces of walnut bark placed on the soil. More soil was added and young tomatoes transplanted to the tub. In four such tubs the tomatoes grew poorly, though they did not die. In two other tubs containing the same type of soil but no walnut root bark, the tomatoes

grew normally. In a third experiment, soil was removed from the area in which the tomatoes shown in figure 4 were planted. This soil was taken at different intervals from the trunk of the tree and placed in separate large tubs. The intervals were 10, 12, 25, 50 and 80 feet from the trunk of the tree. None of the tomatoes planted in these tubs showed any evidence of toxicity from the soil, and one would conclude that there is no toxic substance generally distributed in the soil around walnut trees but that it is localized within the vicinity of the walnut roots.

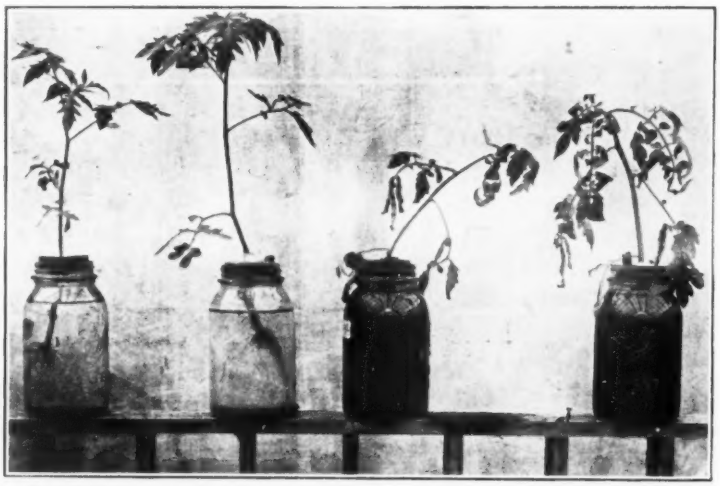


FIG. 5. Wilting of tomato plants in water culture. The two cultures to the left are checks in which no walnut bark was placed. The two jars showing wilting plants contained the same solution as the checks but with walnut root bark added.

Observations and experiments here reported indicate clearly that there is an antogonistic action of walnut roots which proves fatal to some plants. The action is evidently one of toxictiy. However, there is little or no poisoning of the soil as the roots of the affected plants must always be in close contact with those of the walnut. The toxicity is quickly apparent in water cultures of tomatoes containing walnut root bark, and slower in development where walnut root bark is incorporated in soil. The roots of the affected plant become brown in color and die. The tops die, not through direct action but because the roots cease to function.

The toxic principle, it would seem, is either insoluble in the soil water or it undergoes some chemical change shortly after leaving the walnut root, thereby losing its toxicity. Inasmuch as the roots of the affected plant are always in close contact with those of the walnut, there may be no secretion

of the toxic principle by the walnut but it may be taken from the walnut by the action of the other roots.

Considering the constituents of the walnuts that may give rise to the toxic action, the substance juglone seems to be the most likely one, inasmuch as it is a naphthaquinone.

The quinones are known to have marked physiological action. Benzoquinone as a bacteriocide is 160 times more efficient than phenol in its action against *Bacillus typhosus*, (6). In a preliminary experiment containing two controls, the writer has found tomato cuttings in distilled water to be injured by benzoquinone in a concentration of 10 parts per million. Schreiner and Reed (4), in discussing the toxic action of several organic compounds upon wheat, state:

> "Chinone (Benzoquinone) is one of the most toxic compounds whose effect upon wheat seedlings was studied. A concentration of 100 ppm. was fatal in nine days and even 1 ppm. produced an injurious effect. In the intermediate concentrations the chinone was correspondingly injurious. The plants scarcely survived in a concentration of 50 and 25 ppm., and only in concentrations of 10 and 1 ppm. was there any growth comparable to that of the controls in distilled water.
>
> "The great toxicity of chinone is probably due to two of its chemical properties. The first of these is its strong oxidizing power, by virtue of which it is probably able to oxidize labile compounds which exist in cells of the plants and render them unsuitable for use in metabolism. The second property which gives chinone a toxic action is its ketone nature. Chinone readily forms bromin addition products. It united with one molecule of hydroxylamin to form chinon-oxime; with two molecules of hydroxylamin to form chinondioxime. The ketones, as is well known, are distinctly toxic to plants, and taken together these two properties undoubtedly account for the action of chinone upon plants."

The chemical properties of juglone are very similar; it forms mono- and dioxime and bromin addition products (7). It is an irritant causing violent sneezing and has been found, in comparison with benzo-quinone, to be especially valuable as a medicant for skin diseases. It is very slightly soluble in water but more so in organic acids. In dilute alkalin solution it undergoes oxidation.

On account of the difficulty of obtaining the pure substance, no experiments have been carried out to study the effect of this material on plants. It is hoped to do this in the near future.

SUMMARY

1. Walnut (*Juglans nigra* and *Juglans cinerea*) has an antagonistic action which causes a wilting and dying of certain plants such as alfalfa, tomato, and potato.

2. Roots of the affected plants were always in close contact with walnut roots; the toxic substance is not generally distributed in the soil around walnut trees, but is localized in the vicinity of the walnut roots.

3. Walnut root bark contains a substance which is toxic to the roots of tomato plants grown in water culture.

4. It is likely that juglone, or some similar substance, is the toxic constituent of the walnut.

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BLACKSBURG, VIRGINIA

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PHYSIOLOGIC SPECIALIZATION AND MUTATION IN HELMINTHOSPORIUM SATIVUM[1]

J. J. CHRISTENSEN[2]

WITH FOUR FIGURES IN THE TEXT

INTRODUCTION

Helminthosporium sativum P. K. and B. has been studied extensively in recent years. It is an extremely interesting and important pathogene. It causes seedling blight, root rots, basal stem rot, spike and seed blight, leaf spots, stem lesions, and premature death of wheat, barley, rye and many grasses. Although the pathogene has been investigated extensively, there have been many conflicting statements regarding its morphological and physiological characters and its pathogenicity. These conflicting results can be explained very largely by the fact that *H. sativum* is a group species of many physiological forms which mutate readily. The writer has studied thirty-seven forms in detail. A knowledge of the number, cultural characteristics, parasitic capabilities, and genetic stability of these forms is prerequisite to a proper understanding of their pathogenicity and is essential to the plant breeder in his endeavor to develop resistant varieties of grain. Just as important is it to know how frequently these forms mutate, the behavior of the mutants in culture, and their effect on the host.

OBJECTS OF THE INVESTIGATION

The objects of the investigation were: (1) to ascertain the number of physiologic forms; (2) to ascertain the differences in virulence or pathogenicity of the different forms and their mutants; and (3) to determine the degree of stability of these forms.

METHODS AND MATERIALS

All physiologic forms used in the following studies were derived from single spore isolations. The method of procedure was as follows: A mass of spores was taken from the culture with a platinum wire and mixed with 15–20 cc. of sterile water in a test tube. About 0.5 cc. of this spore suspension was poured into a tube of melted agar. Usually two more successive cultures were made in a similar manner. The agar from each tube was

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[2] The writer gratefully acknowledges the helpful advice and criticism of Dr. E. C. Stakman, under whose direction the work was done.

poured into a sterile petri dish, 10 cm. in diameter, and incubated at room temperature. From 12 to 24 hours after the spores had germinated, the plates were examined. Germinating spores, sufficiently far apart, were marked by a circle of India ink on the petri dish. The marked area was carefully examined as to purity from germinating bits of mycelium and germ tubes of other spores before they were cut out and put into separate tubes of agar. The piece of agar containing the germinating spore was first placed on the side of the tube and examined to ensure that only one spore was introduced into the new medium.

The seeds of cereals were obtained from the United States Department of Agriculture and from the Agronomy Division, University of Minnesota. All seeds used in pathogenicity experiments were treated with Jensen's modified hot water treatment or with Chlorophol, an organic mercury compound, in order to kill, in so far as possible, intraseminal mycelium.

The soil used in the experiments was a mixture of three parts garden loam and two parts sand. All the soil for a given experiment was mixed in one lot and then steamed for three hours. The inoculum was grown in Erlenmeyer flasks on autoclaved seed of wheat and oats, in a proportion of three to one by volume. Even the forms that grew slowly on ordinary culture media grew well on this medium. An equal amount of inoculum was added to each pot of a series. The same quantity of uninoculated wheat and oats mixture was added to the controls. The pots were placed on a center bench in the greenhouse in order that all might receive the same amount of heat and light.

EXPERIMENTAL RESULTS

Cultural Characteristics. More than fifty forms were studied, thirty-seven of them in detail. In order to compare their cultural characters, the thirty-seven forms were grown on two different media: first, a one per cent potato dextrose agar; second, a mixture of oatmeal, rice, and cornmeal. Some forms were grown on other media also. Triplicate plates of each medium were inoculated with each form. Small, and as nearly as possible equal, portions of medium containing mycelium, and usually spores, were used as inoculum in each case. The plates were of uniform size and contained the same amount of medium, usually 18 cubic centimeters. The agar was made in one lot, tubed, sterilized, and poured at the same time. After inoculation the plates were placed on the same table in the laboratory and kept at room temperature. Thus all were subjected to the same general environmental conditions. The triplicate colonies of the same forms on the same medium were always alike, unless contaminated by other organisms. It was noticed that a small colony of bacteria sometimes profoundly affected

the growth of certain forms. In the immediate vicinity of colonies of a foreign organism, sporulation was often stimulated, and the morphology of the mycelium was occasionally changed considerably.

The thirty-seven forms studied can be differentiated on culture media macroscopically by the following characters: rate of radial growth; relative amounts of submerged and aerial mycelium; nature of mycelial growth, whether woolly, or cottony, etc.; zonation, whether lacking, prominent, moderate, or faint, and frequence of zones and distance apart; conidial production, whether absent, scarce, moderate, or abundant; conidial clusters; and color of mycelium, white to black with intervening gradation and tints of other colors. The length, width, shape, and septation of the conidia also are different in some of the forms.

The cultural characters of many of the forms of *H. sativum* differ greatly from each other on the same medium. The cultural characters of the same forms are also different on different media. The colonies of many forms were so strikingly different in general appearance on a given medium that one was prone to separate them into different species. However, their similarity on other media forbade such a reclassification.

FIG. 1. Seedlings of Marquis wheat grown in soil inoculated at time of planting with *H. sativum*, showing the comparative virulence of different forms of the pathogene.

A Control
B Form 26
C Form 21
D Form 3
E Form 19
F Form 22
G Form 8
H Form 5

Pathogenicity of Physiologic Forms. Christensen (5), in 1921, noted variations in the degree of pathogenicity of certain physiologic forms of *H. sativum*. Dosdall (6) found a difference in degree of severity with which two strains of *H. sativum* attacked Lion barley and Marquis wheat. Henry (8) observed variation in virulence of strains of *H. sativum* obtained from different sources and was able to distinguish four strains of "small-spored Helminthosporia" by their differences in pathogenicity on wheat.

The writer made tests of the comparative virulence of physiologic forms by inoculating soil in four 4-inch pots with pure cultures. Twenty-five seeds were planted in each pot, so that one hundred seeds of each cereal variety were used in each test.

The comparative pathogenicity of twenty-six forms was determined on Marquis wheat (C. I. 3641) and on Mindum (Minn. No. 470), a durum variety. Both varieties were more or less susceptible to all twenty-six forms of the pathogene, but there were distinct and consistent differences in the virulence of different forms on both varieties of wheat. Forms 3, 19, 21, and 26 were relatively weak (Fig. 1). There also were intermediates between the two extremes.

Differences also exist in the virulence of the different forms on varieties of barley. Trebi (C. I. 936) and Chevalier (C. I. 278) were inoculated with all the thirty-seven forms. Many of the physiologic forms differ in their reaction on two varieties of barley. Forms 5, 11, 34, and 37 were especially virulent on both varieties. Two of these forms, 5 and 11, were virulent on wheat also. Although Forms 3, 4, 30 and others were weakly parasitic, they all attacked barley slightly. In general, the same forms were also weak pathogenes on wheat varieties.

Stability of the Physiologic Forms. The range of variability of *H. sativum* is very wide. The character and rate of growth, the ability to reproduce, and the morphology of the organism are influenced profoundly by the kind, proportion, and amount of foods available and by other environmental conditions. Dosdall and Christensen (7), Stevens (9), and others have demonstrated this conclusively. Besides, it has been shown that there are many forms of *H. sativum* which respond differently to various ecological conditions.

Temporary phenotypes, or modifications, induced by differences in environmental conditions, occur frequently, although the genotype remains unaltered. Such variants revert to the parental phenotype as soon as the causal stimulus is removed. Comparative cultural tests carried on for several years indicate that some forms of *H. sativum* remain constant and always appear the same under identical conditions. The question then arises, how did the forms originate and are they relatively stable? New forms of life are generally supposed to arise as a result of hybridization or mutation.

Recent publications (1, 2, and 9) indicate that asexual mutations in fungi occur frequently on culture media. In 1922, Stevens (9) presented evidence to show that mutations were common in *Helminthosporium* growing in culture. In the present studies much of Stevens' work was repeated and extended, and in general the results were corroborated.

Numerous and widely different types of mutants, occurring usually in sectors, have been observed repeatedly. These variations arose from parts

of mycelium and were generally wedge-shaped or fan-like in shape (Figs. 2 and 3). Under certain conditions, mass-like mutation apparently occurred in some forms. This type of mutation recently has been described also in higher plants (3).

In the present work, numerous transfers were made from sectors and normal parent material. When the original variant was chosen from a sharply defined sector of a relatively young culture, as illustrated in Fig. 2, it always developed into a colony distinct from its parent. Furthermore,

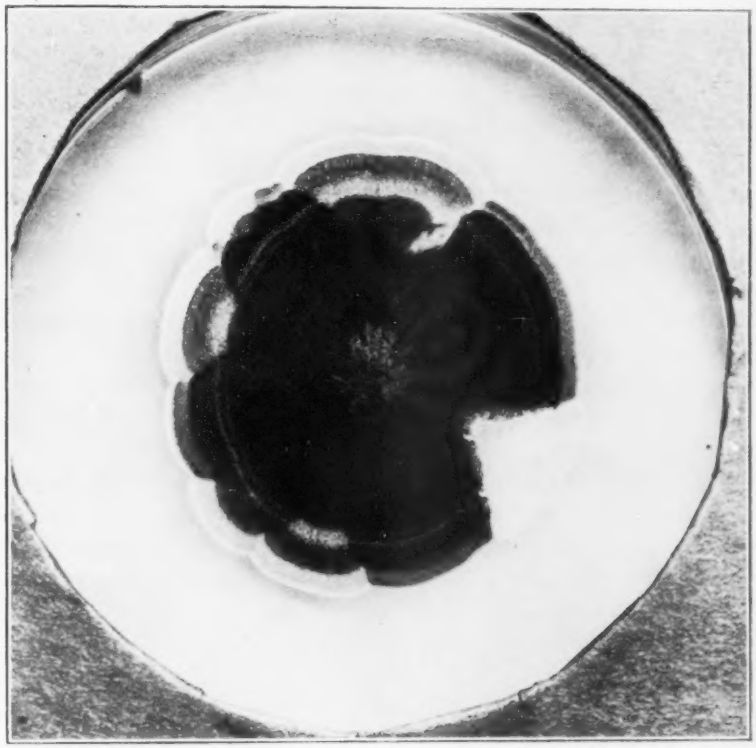

FIG. 2. *H. sativum*, Form 1, on oatmeal-rice-cornmeal agar, showing mutants.

a study of subsequent transfers from these sectors indicated that the changes were genotypic and not merely changes in phenotypes due to environmental conditions. Single-spore isolations, whether from the sector in which a variant originated, or from colonies developed from mass transfers, made similar growth on culture media. In either case the colonies were all alike. Mutants bred true when propagated from spores or mycelium. Stevens (9), Burger (4), and others also have demonstrated that spores from mutants again produced the mutant type.

In order to ascertain whether there were any differences in the tendency for various forms to mutate, two kinds of media were inoculated. Each form was grown in triplicate plates of uniform size, containing 18 cc. of the medium. The plates for each series were inoculated on the same date and kept on the same table in the laboratory.

Some forms mutate more often than others. A given form may mutate more frequently on one nutrient medium than on another. On potato dextrose agar, eighteen out of thirty-seven forms developed sectors or apparent mutations, while sectors occurred in cultures of twenty-seven of the thirty-seven forms when grown on oatmeal-rice-cornmeal agar. Seven forms failed to develop sectors on either medium. Forms 26, 29, and 37 gave rise to apparent mutations on potato dextrose agar but none on the other medium. Twelve of the forms which did not mutate on potato dextrose agar did so on the oatmeal-rice-cornmeal agar. The differences in the number of sectors of certain forms on the two media were sometimes very marked. Thus, Form 33 developed thirty-six; Form 30, fifteen; Form 16, eight sectors on oatmeal-rice-cornmeal agar but on potato dextrose agar none, five, and one, respectively. Mutations occurred in forms obtained from England, Australia, Africa, Argentina, Serbia, and Canada, and from various localities in the United States.

Some of these mutants have been grown on several different media for varying lengths of time, but they have remained permanent for the characters under observation. Mutants appeared as stable as the various forms of *H. sativum* which have been isolated from naturally infected plants. Although it is true that several mutants apparently reverted, these reversions were always in the form of a sector. This agrees with the observations of Stevens (9). Temporary modifications, such as sometimes appear—especially in old cultures—and which revert to the parental type on the first transfer, are not considered here.

Some of the mutants which were cultured mutated still further. Colonies of a mutant have been observed to produce distinct sectors which in turn mutated while still in the same plate. Two mutants gave rise constantly to new mutants, their action suggesting genetic contamination.

In order to determine the effect of the amount of medium on the expression of variation, the following test was made. Newly poured agar plates were placed on a slight incline so that the upper side was covered by a thin layer of the medium, the lower by a considerably thicker layer. These slants were inoculated with Form 1 in the center in the usual manner. Numerous sectors developed, but they developed only in the region of the thinner layer of agar (Fig. 3). That at least some of these sectors were pure mutants was proved by successive transfers.

The mutants studied varied from the parents in the following characters: (1) rate of growth, (2) nature of growth, (3) conidial production, (4) conidial clusters, (5) mycelial characters, (6) color, and (7) zonation. Many of the gross characters in some forms were quite different from any of those observed in strains or forms isolated from host plants. These mutants differed from each other culturally quite as much as do certain species of *Helminthosporium*, such as *H. teres* Sacc., *H. pedicellatum* Henry, and *H. gramineum* (Rab.).

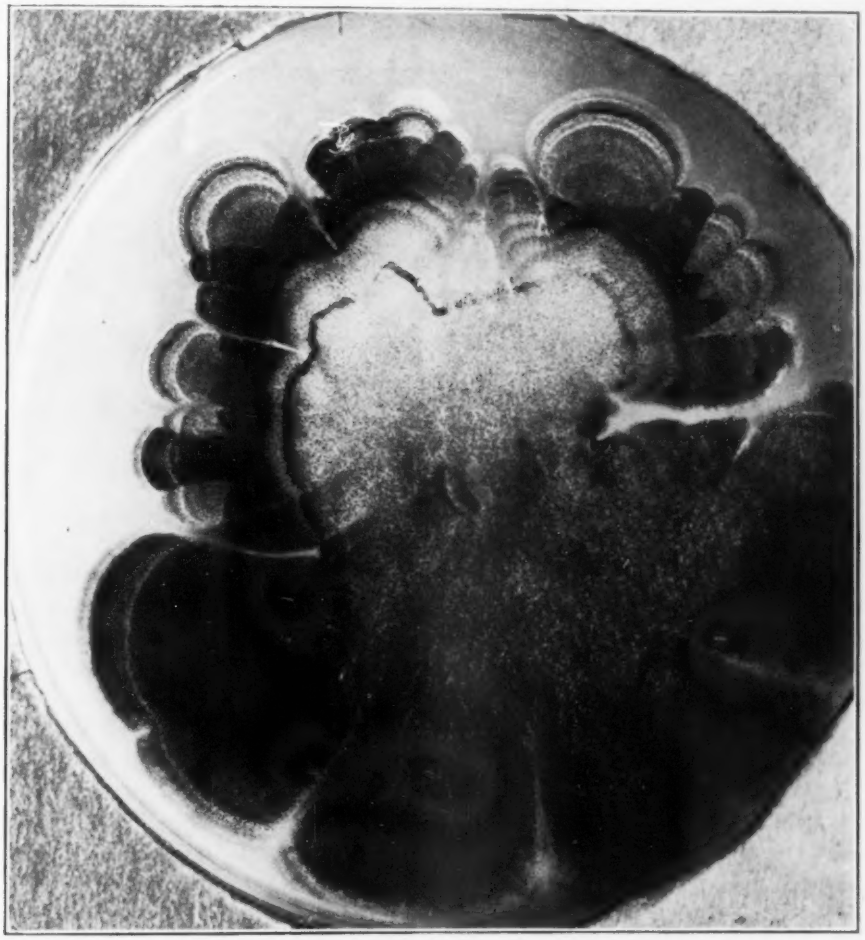

FIG. 3. *H. sativum*, Form 1, on oatmeal-rice-cornmeal agar slant placed so that the depth of agar at the bottom was about three times that at the top. Mutants only on the shallow half of the medium.

Apparently most mutations in fungi have their origin in the loss of a factor, or a group of factors, for color, and in the loss of a factor for high

fructification. Stevens (9) states that mutants with low conidial production, verging on sterility, coupled with paleness of colony, occurred with the greatest frequency. This is in accord with the writer's findings.

Just how these mutations arose is not known. They may have arisen through what one might term normal nuclear rearrangement, or through aberrant chromosomal distribution, or gene changes. It has been impossible to determine accurately the genetic constitution of the parents and their mutants, because the nuclei and chromosomes are very small and the plants are propagated asexually, thus rendering cross-breeding experiments futile. Even if a variant resulted from the fusion of two adjoining cells,

FIG. 4. Seedlings of Marquis wheat grown in soil inoculated with *H. sativum*, Form 22 (back row), and its mutant, No. 40 (front row), showing the difference in virulence of parent and mutant.

it might not necessarily be considered a normal process of combination or segregation, as it has been shown that anastomosing of hyphae is an extremely common phenomenon in *H. sativum*.

Pathogenicity of Mutants Compared with that of their Parents. In order to ascertain whether there are any differences in the degree of virulence of mutants and their parents, comparative tests were made on Trebi barley and Marquis wheat. The experiment was similar to that already described for testing the pathogenicity of the 37 forms of *H. sativum* on barley, except that six pots of each cereal were used. The virulence of thirteen mutants was compared to that of their parents.

All of the mutants did not possess the same degree of virulence. Most

of them were like the parent form, but two were decidedly more virulent than their respective parents. Mutant No. 40 was outstandingly more virulent than its parent, on both barley and wheat (Fig. 4). Three of the mutants were less virulent than their parents.

Mutant No. 40 was derived from a single spore of a monosporous culture of Form 22. It arose as a distinct sector, similar to the one shown in fig. 2, and had been transferred several times and grown on three different nutrient media. The cultural characteristics were not only different from those of its parent, but were unlike those of any of the forms under observation.

DISCUSSION AND CONCLUSIONS

H. sativum is a group species consisting of many physiologic forms. At least thirty-seven can be recognized readily on culture media. The writer observed at least fifty distinct forms, and there are indications of the existence of numerous others. Eleven distinct forms were isolated from material collected in the vicinity of St. Paul. Forms obtained from other workers, or isolated from material collected in different regions, as a rule were different.

The thirty-seven forms studied by the writer differ from each other consistently under the same conditions, and each form is extremely variable under different conditions. For this reason, extreme caution is necessary in describing species such as *H. sativum* and other fungi which may vary to a like degree. At best, a description of *H. sativum* must be based on the characters of relatively few physiologic forms. Not only do the physiologic forms differ in general appearance on culture media, but they are strikingly different physiologically. What is most important, however, they differ pathogenically.

Many of the physiologic forms have quite different parasitic capabilities on wheat and barley. Some forms are extremely virulent, others are moderately virulent, and still others are but weak parasites. These differences in virulence of forms are of paramount importance, as they complicate the study of genetic inheritance and the development of resistant varieties. Varieties which are resistant in one region may be very susceptible in another. Therefore a study of the number and the parasitic capabilities of physiologic forms is prerequisite to sound procedure in breeding work. The problem is essentially local, or, at best, regional. Results obtained in one locality may not be applicable in another.

Mutations occur frequently on culture media. Some forms mutate much more often than others. The mutants observed differed from the parents in rate of growth, color, zonation, amount of aerial mycelium, and patho-

genicity. Relatively weak forms may produce very virulent mutants and *vice versa*. This change in parasitism is tremendously important. *H. sativum* is dynamic, not static. It is continually changing, and consequently presents a continually changing problem. There is every reason to suppose that mutation occurs in nature as well as on artificial media. A final solution of the problem is therefore extremely difficult.

The nuclear phenomena involved in the mutation of *H. sativum* are not known. The possibility of segregation as a result of nuclear fusion is not excluded, although it does not seem to be very strong. Anastomosing hyphae have often been observed. It is barely possible that hybridization may occur quite frequently. The mutants, which arise as sectors, however, suggest that the mechanism is somewhat analogous to bud mutations in higher plants.

SUMMARY

1. There are numerous physiologic forms of *H. sativum*. The writer studied thirty-seven in detail.

2. The thirty-seven physiologic forms can be distinguished in culture by the following characters: rate of growth, relative amounts of submerged and aerial mycelium, nature of mycelial growth, zonation, production of conidia, density of conidial clusters, the color of the mycelium, and the readiness with which the forms mutate.

3. All forms can attack the roots and basal stems of wheat and barley. Some forms are very virulent, while others are relatively non-virulent. In general, there is a correlation between the virulence on wheat and barley.

4. The differences in the pathogenicity of different forms are so great that the conflicting results obtained by previous investigators easily can be explained.

5. Asexual mutation occurs frequently in some forms of *H. sativum*. Mutants arise abundantly from some forms in artificial culture as wedge- or fan-shaped sectors. These mutants bred true when propagated from either spores or mycelium.

6. Some forms mutate readily, others do not. Mutation was observed in forms from England, Australia, Africa, Argentina, Serbia, Canada, and from many localities in the United States.

7. Apparently reversions occur, but, when they do, they are always in the form of sectors.

8. The mutants differ from their parents not only in morphological characters but also in pathogenicity. Some are more virulent than the parents, others are less so.

MINNESOTA AGRICULTURAL EXPERIMENT STATION,
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STUDIES ON THE PATHOGENICITY AND PHYSIOLOGY OF HELMINTHOSPORIUM GRAMINEUM RAB.[1]

THORVALDUR JOHNSON

The barley stripe disease caused by *Helminthosporium gramineum* Rab. has been known for a long time. The pathogene is almost an obligate parasite, and there are practically no authentic records that it has produced conidia on artificial media. Furthermore, the disease has been interesting because the pathogene was supposed to infect plants at flowering time and to cause a systemic infection like that caused by the loose smut fungi. Certain doubts, however, have been raised regarding its exact life history. There seems to be some evidence that the seed or very young seedlings may be infected. There also has been a question as to why the disease should be distributed as it is. It occurs commonly in the more northern barley growing regions, but it is practically unknown in those farther south. The development of the disease differs also in different seasons. The explanation for this seasonal variation might be either differences in temperature or the existence of different physiologic forms. As many of these essential facts regarding *H. gramineum* were obscure, the writer undertook an investigation to elucidate some of the questions.

OBJECTS OF THE INVESTIGATION

The specific objects of the investigation were:

1. To determine the effect of temperature on the severity of infection.
2. To ascertain whether infection would result from inoculating seed or seedlings.
3. To find out whether there are physiologic forms.
4. To determine the conditions most favorable for growth and fructification.

THE RELATION OF TEMPERATURE TO INFECTION

It is well known that early-sown barley usually develops more stripe than that sown at a later date.

Ravn (6), in 1896, made field observations on barley grown from diseased seed planted on May 6 and May 19 respectively. That sown at the former date developed 37.7 per cent of diseased plants, while that sown later produced 21 per cent of infected plants. It therefore appeared likely

[1] Published with the approval of the Director, as Paper No. 560 of the Journal Series of the Minnesota Agricultural Experiment Station.

that a relationship existed between temperature during germination and early growth and infection, but Ravn made no controlled experiments to determine this.

Therefore the writer conducted experiments to ascertain whether such a relationship existed. Seed of Minsturdi barley (Minn. 439), containing naturally infected kernels, was planted in six-inch flower pots (20 kernels per pot) which were placed in soil temperature tanks in the greenhouse at 18–20° C., 23° C., 27° C. and 32° C., eight pots being kept at each temperature. Four pots were kept at from 10–12° C. in a temperature tank regulated by running water. The soil in these pots was covered with a layer of ground cork which served as an insulator between the atmosphere and the soil.

The seedlings were of approximately equal development when removed from the tanks, except in the case of those at 10–12° C., which were smaller when removed. After their removal from the tanks, all the pots were kept under similar conditions in a moderately cool place in the greenhouse.

The first appearance of stripe was observed on January 31, exactly one month after planting, in plants kept at 10–12° C. The average height of the plants in the pots kept at that temperature was then six inches, while those kept at other temperatures were almost ten inches in height. The results are presented in table 1.

TABLE 1.—*The effect of soil temperature on the infection of Minsturdi barley, Minn. 439, by Helminthosporium gramineum*

Soil temp. in degrees C.	Time required for emergence of plants	Time kept in temp. tanks	Number of plants	Plants infected	
				No.	Per cent
10–12	13 days	15 days	75	9	12.
18–20	6 "	9 "	160	7	4.3
23	4 "	7 "	160	1	0.62
27	3 "	7 "	160	1	0.62
32	3 "	7 "	160	1	0.62

The results show: (1) that low temperatures favor infection; (2) that the critical period for infection is during, and immediately after, seed germination.

These results furnish evidence to explain the occurrence of the disease in certain seasons, and its distribution throughout the barley-growing regions of the world. Temperature undoubtedly explains the comparative immunity of barley in warm regions and the severity of the disease in the cooler climates.

SEEDLING INFECTION BY H. GRAMINEUM

Various considerations have led the writer to think that *H. gramineum* might infect seedlings more frequently than is commonly supposed.

Tisdale (8) has shown that *Ustilago tritici* and *U. nuda* may commonly infect seedlings of wheat and barley respectively. Some of the more recent work on *H. gramineum* also lends support to this view. Smith (7), in England, as a result of a re-investigation of the life history of *H. gramineum*, came to the conclusion that Ravn (6) was mistaken in believing that the fungus inhabited the growing point of the host. Smith believes that infection occurs while the shoot is still under the adherent glumes, or during its emergence. The inoculum comprises (a) conidia lodging at the awn end of the grain; (b) mycelium penetrating from the glumes; and (c) perithecia formed inside or outside the glumes. He thinks that the coleoptile, and not the embryo, becomes infected.

Vogt (9) in Germany, in 1923, came to similar conclusions. Histological studies led him to conclude that the mycelium inhabits the space between the glumes and the pericarp of the seed but is never found either in the embryo or the endosperm, and that infection takes place through the coleoptile rather than through the embryo.

Since the time of Von Post, *H. gramineum* has generally been considered to cause "floral infection." While this term definitely limits the time of infection, it does not define the manner of infection nor the tissues of the kernel attacked. In fact, the only thing which the term signifies is that the pathogene is seed-borne. It has been commonly supposed that seedling infection either does not occur or plays only an insignificant part in the life history of the pathogene. Ravn (6) attempted to determine the relative frequency with which the pathogene could attack germinating seedlings. Two hundred and fifty dehulled barley grains of susceptible varieties were germinated in contact with mycelium, with the result that ten plants (4 per cent) became infected. From this Ravn concludes "that stripe disease may be produced by infecting the embryo." He adds, however, that the principal method of infection is by conidia which cause infection during flowering time, forming a mycelium within the seed where "the fungus remains latent with the embryo so long as the grain is dry, and revives when germination commences."

The writer conducted experiments to determine to what extent *H. gramineum* could infect germinating seed. Seed of the susceptible variety Minsturdi (Minn. 439) was used. Ordinary seed and dehulled seed were both inoculated during germination, in various ways, with spores and mycelium of the pathogene. All seed was previously treated by Jensen's hot water treatment, either at 52° C. for 15 minutes or at 46–48° C. for two

hours, in order to kill any mycelium in the seed. Seeds treated with mycelium and spores were sown in six-inch pots kept in temperature tanks at from 18–20° C. The first indications of stripe disease were seen three weeks after planting. Table 2 summarizes the results of the first experiment.

TABLE 2.—*The results of inoculating normal and dehulled seed of Minsturdi barley, Minn. 439, with mycelium and spores of Helminthosporium gramineum*

Kind of seed	Previous hot water treatment	Inoculum	Total number of plants	Plants infected	
				Number	Per cent
Dehulled	52° C. for 15 min.	Spores	44	5	11.3
		Mycelium	34	8	23.5
		Check	20	0	0
Normal	52° C. for 15 min.	Spores	40	0	0
		Mycelium	40	1	2.5
		Check	20	0	0
Normal	46°–48° C. for 2 hours	Spores	40	0	0
		Mycelium	40	0	0
		Check	20	0	0

It is evident from table 2: (a) that infection may occur during germination of the seed; and (b) that the period of greatest susceptibility is prior to the emergence of the coleoptile from the glumes.

Variations in the amount of infection in different experiments may be ascribed to varying technique, as various methods of applying the inoculum were tried.

In the next experiment all seeds were dehulled and soaked in hot water at 52° C. for 15 minutes. Those inoculated with spores were soaked in a spore suspension for a few minutes; others were mixed with mycelium on agar and the soil was also inoculated with mycelium at the time of planting. One-half of the pots were kept at 16° C., the others at 22° C. Table 3 summarizes the results of this experiment.

The first infection was observed nine days after sowing, when the normal seedlings were about four or five inches high. The affected seedlings varied in height from one-half inch to three inches. Most of these early infections resulted in the death of the plants.

An attempt was made to determine the amount of artificial infection which could be induced by inoculating germinating seeds from which the hulls were not removed. Seed was treated in hot water as above at both 46–48° C. and 52° C. Spores and mycelium were both used as inoculum. A spore suspension was poured into melted agar which had been cooled to below 50° C. The seeds were coated with this agar and germinated be-

tween moist blotting papers and finally planted in sterilized soil in six-inch pots. Other seeds were germinated in contact with mycelium on agar in petri dishes and were then planted as above and placed at greenhouse temperatures. The results are summarized in table 4.

TABLE 3.—*Results of inoculating dehulled seeds of Minsturdi barley, Minn. 439, with Helminthosporium gramineum*

Temperature	Inoculum	No. of plants	No. of plants infected	Per cent of plants infected
16° C.	Spores	45	10	22.2
	Mycelium	45	34	75.5
	Check	25	1	4.0
22° C.	Spores	50	16	32.0
	Mycelium	39	23	58.9
	Check	25	0	0

The results were not as conclusive as these figures indicate, because soaking in water at 46–48° C. did not kill all the hyphae in naturally infected seed. Further, the number of seedlings from seed treated with mycelium was reduced, either through the action of the mycelium or the severity of the hot water treatment at 52° C.

TABLE 4.—*Results of inoculating germinating normal seed of Minsturdi barley, Minn. 439, with Helminthosporium gramineum*

Previous hot water treatment	Inoculum	No. of plants	No. of plants infected	Per cent of plants infected
46–48° C. for 2 hrs.	Spores	35	3	8.5
	Mycelium	33	8	24.2
	Check	33	3	9.0
52° C. for 15 mins.	Spores	40	9	22.5
	Mycelium	9	4	44.4
	Check	20	0	0

At any rate, the results indicate that infection of susceptible hulled varieties is possible by inoculating the seed, even though the hulls are not removed. However, it appears more difficult to obtain infection when the hulls are left on the seed than when they are removed.

There has been some difference of opinion among investigators as to the manner in which infection takes place. Ravn (6) states that normally the embryo is infected, and he claims to have histological evidence for the

presence of hyphae in that organ. Vogt (9), as the result of histological studies, claims that neither embryo nor endosperm is infected but that the mycelium is located principally between the glumes and the pericarp of the seed. This view is supported by Smith (7) who is of the opinion that the disease is not truly systemic, as held by Ravn and others.

The fact that it is possible to produce the disease by inoculating germinating seed from which the glumes have not been removed shows that infection may take place through the coleoptile, as the embryo is naturally protected in such seeds. Further work probably is needed to settle definitely the question of the manner of normal infection.

From the point of view of the investigator, the practical value of coleoptile infection by *H. gramineum* is twofold: (1) it affords a means of studying varietal resistance of barley; and (2) it may be used for studying physiologic specialization of the pathogene.

PHYSIOLOGIC SPECIALIZATION

In the light of what Christensen (1) and others have found regarding *H. sativum* P. K. and B., it would not be surprising to find physiologic forms of *H. gramineum*.

A considerable number of single spore isolations were made from material collected at various localities in Minnesota, Colorado, and in Saskatchewan and Alberta, Canada. In investigating the effect of temperature on the growth of the cultures from these isolations, it was found that when they were kept at 32–33° C. for from ten to twelve days and then removed to room temperatures, all the cultures except one recovered and grew normally. The culture in question was obtained from a single spore isolation from infected barley collected at Edmonton, Alberta, Canada. The experiment was repeated several times and similar results were obtained. Evidently this strain is less resistant to high temperatures than the others.

To determine whether the culture was *H. gramineum*, dehulled barley seed was inoculated, and typical stripe disease lesions appeared on the plants.

Morphologically, the mycelium of this strain is not distinguishable from that of the others, nor was any appreciable difference observed in the optimum temperatures of this and other strains. The minimum temperature permitting growth was not determined. The Edmonton strain, however, grew slightly better at from 5 to 6° C. than the others, and it seems clear that its temperature requirements are different from those of the other strains, indicating that it is a distinct physiologic form.

FACTORS INFLUENCING GROWTH AND REPRODUCTION

Helminthosporium gramineum does not readily fructify on artificial media, in this respect differing from other species of *Helminthosporium* which cause cereal diseases. As far as the writer knows, Paxton (5) is the only worker who has reported the production of conidia of *H. gramineum* on artificial media. He obtained perithecia on barley straw and transferred the ascospores to cornmeal agar on which conidia were produced.

The writer attempted to determine the factors influencing the sporulation of *H. gramineum* on artificial media. Several strains were tested, but most of the work was done with a single strain. Persistent attempts to find the right combination of factors for sporulation were made for about seven months, but they were unsuccessful. As the results were negative, it does not seem worth while to discuss the experiment in detail.

The effects of temperature, light, moisture, aeration, hydrogen-ion concentration, various types of media, both solid and liquid, plant tissue extracts, yeast extracts, and numerous variations of nutrients, were tried. The addition of a number of other fungi, and of bacteria, to the culture was also tried with negative results. The addition of certain bacteria resulted in the production of spore-like bodies which, however, failed to germinate. Similarly, the sudden removal of the nutrient solution, coupled with constant light, induced the production of abnormal structures and spore-like bodies, but no normal conidia were formed. On the other hand, variations in aeration, humidity, hydrogen-ion concentration and amount of nutrients resulted only in different degrees of quantitative growth.

In carrying out these experiments, close attention was paid to previous work along similar lines by Klebs (3), Coons (2), Leonian (4) and others, but invariably the methods which they had used successfully with other fungi failed to produce similar results with *H. gramineum*.

The maximum hydrogen-ion concentration which permitted growth was determined as pH 2.52. The limits of hydroxyl-ion concentration were not determined but lie beyond pH 9.25.

The maximum and minimum nutrient concentrations for growth were determined with respect to a number of sugar solutions and found to be respectively $\frac{M}{1}$ and $\frac{M}{1000}$, while optimum growth occurred at from $\frac{M}{10}$ to $\frac{M}{100}$.

SUMMARY

Low soil temperatures were found to favor infection of barley by *H. gramineum* Rab. under controlled experimental conditions. The greatest infection occurred at from 10° C. to 12° C. Very little infection occurred at soil temperatures higher than 20° C.

It has been shown that artificial infection by *H. gramineum*, through inoculations of germinating barley seed, is possible. By removing the hulls it is possible to obtain a high percentage of infection in susceptible varieties. A lower percentage of infection occurs when normal seed of the same varieties is inoculated. The period of greatest susceptibility in dehulled seed appears to be just subsequent to the emergence of the coleoptile.

Evidence has been obtained for physiologic specialization in *H. gramineum*. At least two physiologic forms appear to exist.

Attempts to induce this organism to produce conidia on artificial media were unsuccessful.

The writer wishes to express his appreciation of many helpful suggestions from Dr. E. C. Stakman and Dr. J. J. Christensen. He also wishes to express his gratitude to Dr. Margaret Newton and Prof. W. P. Fraser, of Saskatoon, Sask., Canada, for help in collecting material.

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SECOND REPORT OF PROGRESS ON STUDIES OF CROWNGALL IN RELATION TO NURSERY STOCK[1]

A. J. RIKER AND G. W. KEITT[2]

The writers[3] recently reported that extensive isolation studies had failed to reveal the presence of *Bacterium tumefaciens* Smith and Town. in overgrowths on certain apple trees which had been rejected at nurseries because of supposed crowngall infection. They suggested the working hypotheses: (a) that the malformations dealt with on these trees were not induced by the crowngall organism; (b) that their development was merely incidental to the root-grafting method employed in the propagation of this stock. Further studies have yielded results which are in full conformity with these hypotheses.

The isolation studies previously reported have been extended to 227 apple trees which were representative of lots of stock sent in as crowngall rejects by twelve nurseries in six states. Less than two per cent of these trees yielded *Bact. tumefaciens*. The overgrowths which yielded the crowngall organism were of the "soft gall" type. Tests of the isolation technique on known crowngall at frequent intervals throughout the period of experimentation gave positive results from 44 of 46 plants studied.

A study of the trees which failed to yield *Bact. tumefaciens* showed that most of the malformations developed immediately above places where there was interference with the downward passage of elaborated food. The most common cause of this stoppage was the failure of some part of the cion to unite with the stock. The effects produced appear to be closely comparable with those which follow girdling. Many of the trees, especially those one year old, showed clearly that the failures of union were the result of poor fits in grafting. An examination of representative freshly prepared grafts from seven of the nurseries from which stock had been secured for the

[1] Published with the approval of the Director of the Wisconsin Agricultural Experiment Station.

[2] This work, which is being administered by the Crop Protection Institute, is supported cooperatively by the American Association of Nurserymen and individual nurserymen, the Iowa State College of Agriculture and Mechanic Arts, and the University of Wisconsin, in collaboration with the U. S. Department of Agriculture. Coordinated research programs are in progress at the Iowa State College of Agriculture and Mechanic Arts and at the University of Wisconsin.

[3] Riker, A. J., and G. W. Keitt. A report of progress on studies of crowngall in relation to nursery stock. Science, N. S., 62: 184–185. 1925.

isolation experiments showed ample numbers and types of misfits to account for the abnormalities found. This situation is not surprising in view of the fact that in many nurseries individual workmen cut and fit as many as three thousand grafts a day, an average of one in about twelve seconds of working time.

A study has been made of the types of misfit found in freshly prepared piece-root apple grafts selected at random at certain large nurseries, and plantings have been made for the purpose of following the development of malformations in relation to fit.

Overgrowths similar to those found on the rejected nursery stock studied were produced at will on two-year-old apple trees by certain types of wounding. Likewise, in experiments in which precautions were taken to exclude *Bact. tumefaciens,* malformations about the unions of piece-root apple grafts have been induced or suppressed almost at will by variations in the technique of grafting.

These and other results appear to justify the conclusions: (a) that the major portion of the overgrowths encountered on the piece-root apple grafts thus far studied by the writers, developed incidentally to the grafting process, without the intervention of *Bact. tumefaciens;* and (b) that the occurrence and development of such malformations may be reduced by modifications in grafting practice. It remains for further investigations to determine the relative prevalence of true crowngall and of these non-parasitic overgrowths on apple root grafts under different regional and seasonal conditions, and to show to what extent the latter types of malformation may be controlled in commercial propagation by modifications in grafting practice. Studies bearing on these and related questions are being continued.

UNIVERSITY OF WISCONSIN,
MADISON, WISCONSIN

PHYTOPATHOLOGICAL NOTES

Crown Wart of Alfalfa in Indiana. On June 12, 1925, while examining alfalfa fields near Madison, Indiana, the writer found a number of plants in two fields attacked by what appeared to be crown wart. Microscopical examination of galls from these fields showed that typical fruiting bodies of *Urophlyctis alfalfae* were present in abundance. The one field had been sown some twenty years ago and still had about a fourth of a stand of plants fairly uniformly distributed. No positive evidence was obtained that the disease was causing any damage. However, the season had been very dry and the first cutting had just been made; hence this was not an opportune time for making observations.

As far as the writer has knowledge, this is the first report of the occurrence of this disease east of the Rocky Mountains.—J. L. Weimer, U. S. Dept. of Agriculture, Washington, D. C.

The Avocado Scab Organism. It has been generally accepted that the fungus causing citrus scab also produces the disease of avocado commonly known as avocado scab. Repeated artificial field inoculations on avocado with the citrus scab organism, however, have failed to show infection; while similar inoculations on avocado with the fungus associated with avocado scab have reproduced the disease in all cases. A critical study of the fungus from avocado indicates that it is an undescribed species of the form-genus *Sphaceloma.* A technical description of the new species is being prepared in connection with an account of its pathology.—Anna A. Jenkins, Bureau of Plant Industry, U. S. Department of Agriculture.

Browning Disease of Flax in North America. An important disease of flax, characterized by brown lesions on stems, leaves, and bolls, and a tendency of certain of the diseased stems to break over near the ground line, was described by Lafferty in Ireland in 1921. The former symptoms were referred to as "browning" and the latter as "stem break," and both were shown to be caused by the same organism. Affected stems are brittle, and break during scutching, resulting in shortened fibers; and the yield and quality of the seed also are reduced. The pathogene was described as *Polyspora lini* Lafferty, a new genus and species.[1]

This disease has been present in North America at least since 1920. In August of that year the writer noticed a peculiar spotting on several varie-

[1] Lafferty, H. A. The browning and stem break disease of cultivated flax (*Linum usitatissimum*), caused by *Polyspora lini.* Sci. Proc. Roy. Dub. Soc. n.s. **16**: 248–274. 1921.

ties of flax in the Field Husbandry experimental plots of the University of Saskatchewan, Saskatoon, Saskatchewan, Canada. Specimens were collected, but no attempt was made at that time to isolate the causal organism. In 1923, "stem break" caused by *Polyspora lini* was reported by Fraser[2] as severe in two one-fortieth acre plots at Saskatoon. The writer again collected the disease from varietal plots at Saskatoon in August of the same year, and isolated the causal organism several weeks later. The following fall, *Polyspora lini* was isolated from some of the diseased leaves collected in 1920. These had been kept dry in the laboratory. The fungus had therefore remained alive in this condition for more than four years. Pethybridge *et al*[3] found spores of the fungus to survive on dry flax seed for two and one-half years, but not three years. He points out that infected seed readily transmit the disease to the following crop. The pathogene may therefore be readily introduced into new localities by infected seed.

In August, 1925, specimens of diseased flax were received from Mr. B. B. Robinson, of East Lansing, Michigan. These specimens were collected from a field about 100 miles north of East Lansing, in which about one per cent of the plants were affected. Part of the seed from which this crop was grown was obtained from Ontario, Canada. Two diseases were found on these plants, namely, rust (*Melamspora lini*) and "browning" (*Polyspora lini*). The latter fungus was isolated, and cultures and lesions on the stems compared with authentic material received from Ireland and with that from Saskatoon and found to agree. No previous record of the occurrence of "browning" in the United States has come to the writer's attention.

The symptoms thus far observed have been typical of the "browning" stage of the disease, rather than the breaking over of the plants characteristic of "stem break."

An organism isolated from diseased flax collected at Crookston, Minnesota, and St. Paul, Minnesota, in 1924, resembles *Polyspora lini*, but is not identical with it. It was reisolated from flax which had been artificially inoculated in the field in 1925.—A. W. HENRY, University of Minnesota.

Personals.—Dr. Perley Spaulding, Pathologist of the Office of Investigations in Forest Pathology, Bureau of Plant Industry, Washington, D. C., has been assigned to duty as Pathologist with the Northeastern Forest Experiment Station, at Amherst, Mass. Correspondence and exchanges should be thus addressed.

[2] Survey of the Prevalence of Plant Diseases in the Dominion of Canada. Fourth Annual Report. p. 35. 1923.

[3] Pethybridge, G. H., H. A. Lafferty, and J. G. Rhynehart. Investigations on flax diseases. Jour. Dept. Agr. and Tech. Instruction for Ireland 21: 9. 1921.

Dr. W. D. Moore, Assistant Pathologist in Extension Work, Clemson Agricultural College, South Carolina, has been granted a year's leave of absence and will spend the time as Pathologist for the Truckers Supply Co., Beaufort, South Carolina. He expects to do research work on some of the diseases of truck crops.

Ellsworth Bethel, the well known uredinologist, died at Denver, Colorado, September 8, 1925, aged 62 years.

The Department of Plant Pathology of the Virginia Polytechnic Institute has been expanded to include botany and is now designated as the Department of Botany and Plant Pathology. The Department of Biology, which formerly included botany, has been discontinued, following the resignation and retirement of Dr. E. A. Smyth, Jr., professor of biology. The organization of the enlarged Department is as follows: professor, F. D. Fromme; associate professors, H. S. Stahl (instruction), A. B. Massey (instruction), S. A. Wingard (research); assistant professors, F. J. Schneiderhan (research), James Godkin (extension); instructor, R. H. Hunt (research); assistant, C. N. Priode (research).

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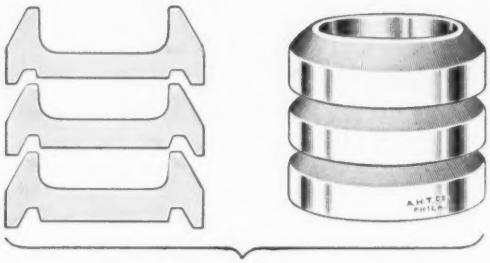

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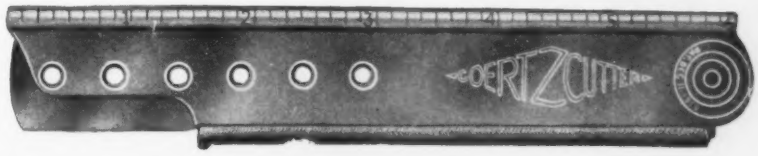

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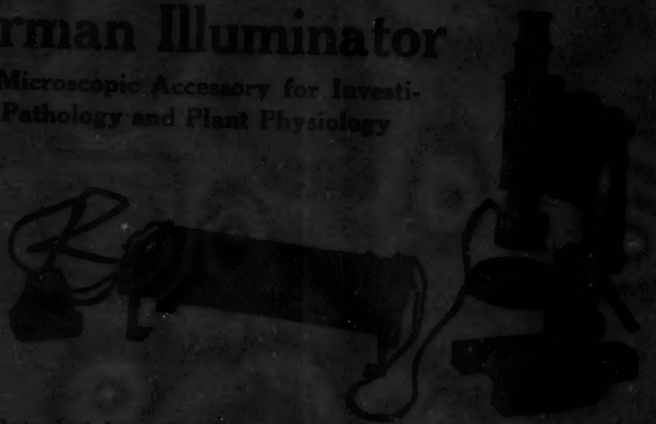

Particularly useful in Botanical Investigations, Plant Pathology and Plant Physiology because the oblique illumination surrounding the objective throws the light exactly where it is wanted. The structural detail of the object under examination in the microscope equipped with a Silverman Illuminator is brought out with astonishing clearness.

You can make better photomicrographs with the Silverman Illuminator, for the light is evenly distributed over the whole field, producing negatives of uniform density.

Can be attached easily to your microscope and will enable you to make investigations heretofore thought impossible.

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